IN THE UNITED STATES DISTRICT COURT
FOR THE MIDDLE DISTRICT OF NORTH CAROLINA

DEMOCRACY NORTH CAROLINA, THE LEAGUE OF WOMEN VOTERS OF NORTH CAROLINA, DONNA PERMAR, JOHN P. CLARK, MARGARET B. CATES, LELIA BENTLEY, REGINA WHITNEY EDWARDS, ROBERT K. PRIDDY II, WALTER HUTCHINS, AND SUSAN SCHAFFER. *Plaintiffs,* *vs.* THE NORTH CAROLINA STATE BOARD OF ELECTIONS; DAMON CIRCOSTA, in his official capacity as CHAIR OF THE STATE BOARD OF ELECTIONS; STELLA ANDERSON, in her official capacity as SECRETARY OF THE STATE BOARD OF ELECTIONS; KEN RAYMOND, in his official capacity as MEMBER OF THE STATE BOARD OF ELECTIONS; JEFF CARMON III, in his official capacity as MEMBER OF THE STATE BOARD OF ELECTIONS; DAVID C. BLACK, in his official capacity as MEMBER OF THE STATE BOARD OF ELECTIONS; THE NORTH CAROLINA DEPARTMENT OF TRANSPORTATION; J. ERIC BOYETTE, in his official capacity as TRANSPORTATION SECRETARY; THE NORTH CAROLINA DEPARTMENT OF HEALTH AND HUMAN SERVICES; MANDY COHEN, in her official capacity as SECRETARY OF HEALTH AND HUMAN SERVICES, *Defendants.*	Civil Action No. 20-cv-457

DECLARATION OF MEGHAN MURRAY IN SUPPORT OF PLAINTIFFS' MOTION FOR PRELIMINARY INJUNCTION

1. I am the Ronda Stryker and William Johnston Professor of Global Health in the Department of Global Health and Social Medicine at the Harvard Medical School, a Professor of Epidemiology at the Harvard Chan School of Public Health, a faculty member of the Center for Communicable Disease Dynamics at the Harvard Chan School of Public Health and an associate Professor of Medicine at the Harvard Medical School and the Brigham and Women's Hospital. I obtained my BA from Dartmouth College in 1980, after which I worked for the Intergovernmental Committee for Migration (now IOM) heading up a public health screening program for refugees being resettled from refugee camps in Thailand. I obtained my MD from Harvard Medical School in 1990 and my ScD (doctorate in science) in Epidemiology from the Harvard School of Public Health in 2001. I completed a residency in internal medicine in 1993 and a fellowship in the sub-specialty of Infectious Diseases in 1995, both at the Massachusetts General Hospital in Boston.

2. Over the past 20 years, I have worked in the field of infectious disease dynamics and epidemiology, teaching and conducting research in emerging infectious diseases and in tuberculosis epidemiology and control. At the Harvard Chan SPH, I taught the basic epidemiology course *Infectious Disease Dynamics* between 2000 and 2016, and I have directly supervised the research of over 40 graduate students and post-doctoral fellows in these fields. Attached here as Exhibit A and incorporated by reference to this declaration is a copy of my curriculum vitae.

3. I have conducted research and have published on the transmission dynamics of SARS-CoV-1 in 2003, the 2010 cholera epidemic in Haiti, and on the 2015 Ebola

outbreak, although most of my research is in the field of tuberculosis. I have published over 200 research articles. My work includes dynamic modeling of epidemics (TB, Cholera, Ebola, SARS-CoV-1, SARS-CoV-2); cohort studies on host and pathogen specific determinants of disease transmission and the development of novel diagnostic tools for the diagnosis of infectious diseases. I have been funded by the National Institute of Infectious Disease and Allergy since 1995 and have led, and currently lead, several major consortium projects on tuberculosis funded by this agency.

4. At the Harvard Medical School, I lead the Global Health Research Core of the Harvard Medical School, which conducts research in more than ten countries on a range of topics including emerging infectious diseases. I head up research at the Division of Global Health Equity at the Brigham and Women's Hospital and also direct research at the non-governmental organization, Partners in Health. I have served as an associate editor of the European Journal of Epidemiology, the Journal of the International Union against TB and Lung Disease and of PLoS (Public Library of Science) Medicine. I am the co-lead of the Epidemiology working group of the Massachusetts Consortium for Pathogen Readiness.

5. I am currently collaborating on research concerning SARS-CoV-2 and its incidence, as well as serving on Covid-19 advisory groups for multiple organizations, including the State of Massachusetts and Harvard University. My research in this area includes, but is not limited to, modeling and estimating the number of hospital beds that will be required in the US and elsewhere, developing methods on syndromic surveillance

for Covid-19 for low and middle-income countries, identification of risk factors for poor outcomes and the use of the vaccine, BCG, to prevent Covid-19 disease. To date (May 11, 2020), I have published two papers in this area and have three others under review.

OVERVIEW

6. **SARS-CoV-2 is a newly identified coronavirus that is the causative agent involved in Coronavirus Disease 2019 (Covid-19). SARS-CoV-2 infection can result in an asymptomatic infection or in symptomatic disease which ranges from mild to severe.** Most people who develop symptomatic Covid-19 have a flu-like illness that starts out with fever, cough, sore throat and shortness of breath. A subset of people who are infected will go on to develop much more serious illness, characterized by respiratory compromise and acute respiratory distress syndrome (ARDS). Other serious manifestations of Covid-19 have included cardiac problems: arrhythmias, acute cardiac injury, and shock.

7. Because Covid-19 is a new disease, it is too early to know the full extent of long-term medical consequences of the infection. However, some information can be inferred from the courses of diseases with similar manifestations. Patients who develop ARDS and/or are mechanically ventilated are likely to develop lung scarring that may permanently impair their pulmonary function [1]. Patients who end up in ICUs or on mechanical ventilation for extended periods often develop post-ICU syndrome which prolonged physical debilitation, muscle atrophy, neurocognitive impairments and emotional/psychiatric responses that are similar to post-traumatic stress syndrome.

Although Covid-19 has been reported in people of all ages, older people and those with co-morbidities (concurrent illnesses) are most likely to develop severe disease.

8. **Covid-19 is a respiratory virus which is spread by symptomatic and asymptomatic people through respiratory droplets, meaning drops of fluid from the nose or mouth that are emitted during coughs, sneezes or even talking.** Some of the viral particles emitted this way end up on surfaces (door handles, coins) where they can remain viable. It has also been shown that Covid-19 can be transmitted as an aerosol – in other words, through the airborne route, *i.e.*, direct inhalation of virus suspended in the air.

9. **Control of SARS-CoV-2 spread is particularly difficult relative to some other viral infections because people can transmit the infection even when they do not have symptoms of the disease.** This means that the practice of isolating patients with symptomatic disease will not be enough by itself to control epidemic spread. In contrast, infections like smallpox and SARS-CoV-1 were not infectious until symptoms had developed so isolation of ill people had a substantial impact on epidemic control. In the absence of a vaccine or pharmaceutical interventions that interrupt transmission, infection control can only be achieved by reducing the number of contacts between infectious individuals (including those who are asymptomatic) and susceptible people.

10. Infectious disease epidemiologists have developed projections of the future trajectory of Covid-19 incidence based on modeling the epidemic and possible interventions. Although these models differ in terms of specifics, they consistently show that it is highly likely that the relaxation of social distancing measures that will occur with

the end of "lock-down" will increase the number of social contacts that people make and that the incidence of infection will increase accordingly In particular, these models predict that transmission of SARS-CoV-2 will continue or increase in the fall and winter, leading to further morbidity and mortality from this disease.

11. There is a substantial risk that an infection with Covid-19 acquired during voting at a poll booth in North Carolina in the fall of 2020 could result in symptomatic disease, hospitalization or death. The risk of an individual being infected during voting at a polling booth in fall 2020 depends on the number of infectious people in that community at that time point and the number of physical, fomite-mediated and near contacts one makes during that process. To the extent that polling places are crowded, require people to wait in lines, involve interacting with polling staff or other voters at a close distance, move people through the process slowly, are poorly ventilated, and/or involve people touching objects like pens, paper, or surfaces within the voting booth, they constitute a risk to voters. Similarly, if voters or poll workers use toilets that are also used by others, they can be put at risk. North Carolina has relatively high rates of the co-morbidities that predispose people to poor outcomes from Covid-19.

12. ***I was asked to describe the novel coronavirus that causes Covid-19.*** SARS-CoV-2 is a newly identified coronavirus that is the causative agent involved in Coronavirus Disease 2019 (Covid-19) [1]. It is a single-stranded RNA virus of the Coronavirus family. Previously identified coronaviruses are known to infect a wide range of hosts including wild and domestic animals and birds as well as humans. Six human coronaviruses have

been identified over the past 60 years; four of them (OC43, 229E, NL63, and HKU1) cause mild cold-like symptoms and/or gastrointestinal tract infections. Two that have caused more serious illness include the severe acute respiratory syndrome coronavirus (SARS-CoV-1) that emerged in China in 2003 and the Middle East respiratory syndrome coronavirus (MERS-CoV) that was first identified in humans in Saudi Arabia in 2012. SARS- and MERS-CoVs are believed to have originated in bats and transferred to humans through intermediary hosts, possibly palm civets for SARS or dromedary camels for MERS. The coronaviruses that are most similar to SARS-CoV-2 are those identified in horseshoe bats – these share 96 percent of their genetic material with SARS-CoV-2 while the earlier SARS virus shared 80 percent and cold viruses mentioned above share about 50 percent [2].

13. Like SARS-CoV-1, SARS-CoV-2 infiltrates human cells by binding to the receptor for ACE2 (angiotensin converting enzyme) and then being taken up by these cells, where it directs the production of new virus particles (virions) using the host's genetic machinery [3]. Like other viruses, SARS-CoV-2 virions consist of a "core" which contains the genetic material, a "capsid" which is a protein coat and a lipid envelope. Upon assembly in the host cell, newly-produced virions are released from the host cell and go on to infect new host cells. To some extent, the clinical manifestations of the disease are related to the types of cells that have the receptor to which the virus binds and to the inflammatory responses that are induced by the host immune response to the infection. While ACE2 receptors were well-known to be present on vascular endothelial cells (blood vessels) and

renal tubular cells (kidney), they have also been found to be abundant on alveolar epithelial cells (lung), enterocytes (gut), heart cells, brain cells and in cells in the inner lining of the nose [4]. This diverse distribution helps explain the wide constellation of symptoms and syndromes that are increasingly being recognized as part of Covid-19 disease.

14. ***I was asked to characterize clinical features of Covid-19.*** SARS-CoV-2 infection can result in an asymptomatic infection or in symptomatic disease which ranges from mild to severe. The term Covid-19 refers to the illness that is caused by SARS-CoV-2. Most people who develop symptomatic Covid-19 have a flu-like illness that starts out with fever, cough, sore throat and shortness of breath. As clinicians have gained more experience with the disease, it is now becoming clear that the initial presentation of the disease can also include a variety of other symptoms including gastrointestinal issues such as nausea, vomiting and diarrhea, loss of a sense of taste and/or smell, headache and muscle pain and in some cases, particularly in the elderly, altered neurological states such as confusion, lethargy and reduced responsiveness. The Centers for Disease Control and Prevention (CDC) have recently expanded their list of symptoms associated with Covid-19 from fever, shortness of breath and cough to include chills, muscle pain, headache, sore throat and new loss of taste or smell [5]. On average, among those who present with these symptoms, fever persists for around 12 days, shortness of breath for 13 days and cough for about 19 days. According to the World Health Organization (WHO), recovery time appears to be around two weeks for mild infections and three to six weeks for severe disease [6].

15. A subset of people who are infected will go on to develop much more serious illness, characterized by respiratory compromise due to pneumonia that can be gradual or sudden. Some patients who initially reported only mild symptoms may progress to severe disease over the course of a week. In one study of 138 patients hospitalized in Wuhan, China, for pneumonia due to SARS-CoV-2, dyspnea (severe shortness of breath) developed approximately five days after the onset of symptoms, and hospital admission occurred after around seven days after the onset of symptoms [7].

16. Acute respiratory distress syndrome (ARDS) is the major complication in patients with severe disease. In the study cited above, ARDS developed in 20 percent of hospitalized patients around eight days after the onset of symptoms and 12.3 percent of this group required mechanical ventilation [7]. In another study of 201 hospitalized patients with Covid-19 in Wuhan, 41 percent developed ARDS [8]. Some patients with severe Covid-19 have an overactive inflammatory response, sometimes termed a "cytokine storm," which is characterized by persistent fevers and laboratory abnormalities including high levels of inflammatory markers and elevated proinflammatory cytokines. People with these types of laboratory abnormalities are those most likely to have critical or fatal illness.

17. Other serious manifestations of Covid-19 have included cardiac problems: arrhythmias, acute cardiac injury, and shock [9-11] which occurred in 17, 7, and 9 percent of hospitalized patients, respectively [7]. In a case series of 21 severely ill patients admitted to a US ICU, one-third developed cardiomyopathy (injury to the heart muscle) [12]. An alarming recent finding has been the association of Covid-19 with thromboembolic

complications (pulmonary embolism and stroke) that have been reported among patients in younger age groups and without known risk factors [13-15]. In one US-based case series, a single health facility reported on five Covid-19 patients with acute stroke who were seen over a two-week period; all of these people were under 50 years of age [14]. This incidence is more than seven times the rate reported in that age group prior to the pandemic. In one series of ICU patients, ischemic stroke was also noted observed in 3.7 percent of the patients [15].

18. Other rarer manifestations of Covid-19 include Guillain-Barré syndrome which can occur five to ten days after initial symptoms [16]. Guillain-Barré syndrome is a rare neurological syndrome characterized by an inflammation of nerve cells outside the brain. In serious cases, it can lead to paralysis which usually resolves after six months but which can be permanent in some cases. Another rare inflammatory syndrome that has been reported in Covid-19 occurs in children who have developed symptoms consistent with toxic shock syndrome and Kawasaki disease [17].

19. Because Covid-19 is a new disease, it is too early to know the full extent of long-term medical consequences of the infection. However, some information is already available, and some can be inferred from the courses of diseases with similar manifestations. Patients who develop ARDS and/or are mechanically ventilated are likely to develop lung scarring that may permanently impair their pulmonary function. [18]. In addition, patients who end up in ICUs or on mechanical ventilation for extended periods often develop post-ICU syndrome which includes a constellation of findings such as

prolonged physical debilitation, muscle atrophy, neurocognitive impairments and emotional/psychiatric responses that are similar to post-traumatic stress syndrome [19]. Patients that suffer strokes in the context of Covid-19 are very likely to experience long-term neurological deficits from these events.

20. Although Covid-19 has been reported in people of all ages, older people and those with co-morbidities (concurrent illnesses) are most likely to develop severe disease. Accurate case fatality rates are hard to obtain in the context of limited testing since we do not always know who actually has the infection. However, a compilation of the death rates across countries shows that older people are consistently more likely to die if they have detectable Covid-19 disease than are younger people [20]. The table below shows that the risk of death rises with each additional decade after age 50.

Table 1. Case Fatality rate by age groups. From Onder G, Rezza G, Brusaferro S. Case-Fatality Rate and Characteristics of Patients Dying in Relation to Covid-19 in Italy. *JAMA.* 2020;323 [18].

Table. Case-Fatality Rate by Age Group in Italy and China[a]

	Italy as of March 17, 2020		China as of February 11, 2020	
	No. of deaths (% of total)	Case-fatality rate, %[b]	No. of deaths (% of total)	Case-fatality rate, %[b]
All	1625 (100)	7.2	1023 (100)	2.3
Age groups, y				
0-9	0	0	0	0
10-19	0	0	1 (0.1)	0.2
20-29	0	0	7 (0.7)	0.2
30-39	4 (0.3)	0.3	18 (1.8)	0.2
40-49	10 (0.6)	0.4	38 (3.7)	0.4
50-59	43 (2.7)	1.0	130 (12.7)	1.3
60-69	139 (8.6)	3.5	309 (30.2)	3.6
70-79	578 (35.6)	12.8	312 (30.5)	8.0
≥80	850 (52.3)	20.2	208 (20.3)	14.8

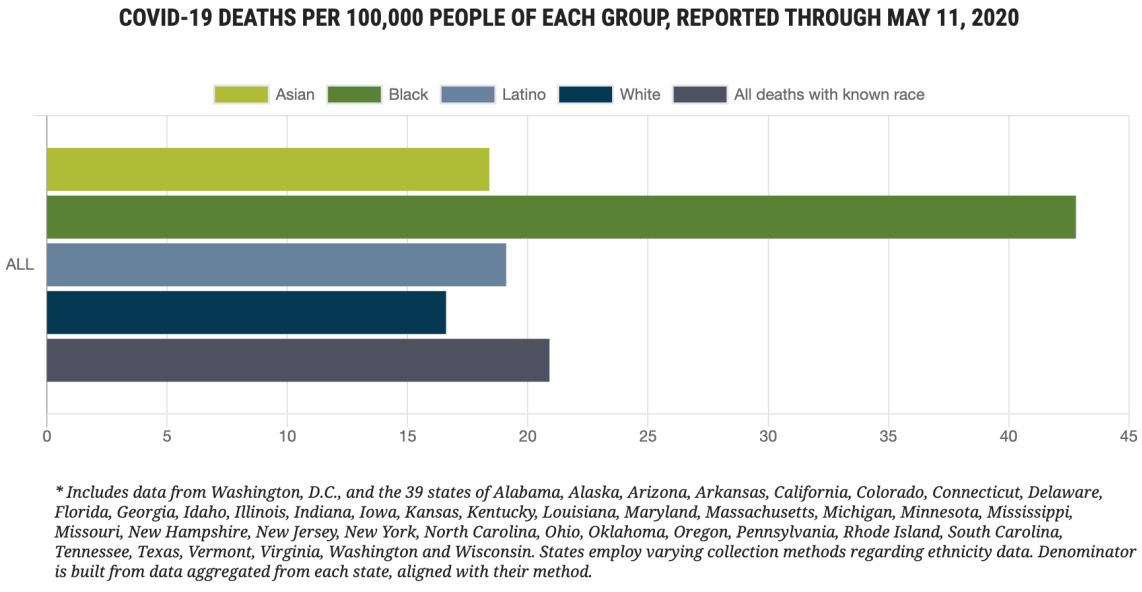
[a] Data from China are from Chinese Center for Disease Control and Prevention.[4] Age was not available for 1 patient.

[b] Case-fatality rate calculated as number of deaths/number of cases.

21. In addition to age, other risk factors for severe disease and death include hypertension, heart disease, lung diseases (e.g., asthma, chronic obstructive pulmonary

disease (COPD)), diabetes mellitus, obesity, and chronic kidney disease. In one recent study of 5700 Covid-19 patients identified in New York City, 56.6 percent had pre-existing hypertension, 41.7 percent were obese (body mass index > 30) and 33.8 percent had diabetes mellitus [21]. Risk factors for death among patients with Covid-19 were recently ascertained in another study of 5,683 Covid-19 deaths in the United Kingdom [22]. In this report, men were twice as likely to die as women; people with obesity 2.3 times as likely to die as those of normal weight; people with uncontrolled diabetes 2.36 times than non-diabetics, people with organ transplants 4.3 times than their healthy counterparts. In both Britain and the US, there are marked disparities in deaths by race: 33 to 42 percent of deaths in the US have reportedly occurred in African Americans, while only 12 to 13 percent of the total US population is African American [23]. Figure 2 provides comparative death rates from Covid-19 from the APM research lab (https://www.apmresearchlab.org/).

Table 2 Covid-19 Death Rate by ethnic group. (From APM Research Lab)
https://www.apmresearchlab.org/



** Includes data from Washington, D.C., and the 39 states of Alabama, Alaska, Arizona, Arkansas, California, Colorado, Connecticut, Delaware, Florida, Georgia, Idaho, Illinois, Indiana, Iowa, Kansas, Kentucky, Louisiana, Maryland, Massachusetts, Michigan, Minnesota, Mississippi, Missouri, New Hampshire, New Jersey, New York, North Carolina, Ohio, Oklahoma, Oregon, Pennsylvania, Rhode Island, South Carolina, Tennessee, Texas, Vermont, Virginia, Washington and Wisconsin. States employ varying collection methods regarding ethnicity data. Denominator is built from data aggregated from each state, aligned with their method.*

22. Although the Covid-19 case fatality rates are low in young individuals, it is important to note that multiple seroprevalence studies (studies that detect previous infection in people) in several countries show that infection (as distinct from severe disease) is more common in people younger than 50, probably because they have more frequent social contacts than older people. Furthermore, the proportion of people in the US population under 50 years of age is 66%, meaning that even though the absolute risk for a young person is lower than for someone over 50, deaths among people under 50 will not be uncommon as the epidemic progresses over time.

23. ***I was asked to explain how Covid-19 is transmitted and to describe interventions that could interrupt transmission.*** SARS-CoV-2 can be transmitted in multiple ways, through respiratory droplets emitted during talking, singing, sneezing and coughing, via objects on which viral particles have been deposited, and through air. Importantly, the virus can be transmitted by people who are asymptomatic as well as by those who are demonstrably ill.

24. Covid-19 is a respiratory virus which is spread through respiratory droplets, meaning drops of fluid from the nose or mouth that are emitted during coughs, sneezes or even talking [52]. Some of the viral particles emitted this way end up on surfaces (door handles, coins) where they can remain viable. These objects then become "fomites," defined as inanimate objects that can transfer infection between people. A recent study documented the stability of SARS-CoV-2 on a series of different surfaces over time [24]. The virus was found to be more stable on plastic and stainless steel than on copper and

cardboard with viable virus detectable for up to 72 hours after application to these surfaces although the virus titer was steadily reduced over those periods. On cardboard, viable SARS-CoV-2 was measured for 24 hours. Notably, this study also evaluated the stability of SARS-CoV-1 – the causative virus of the 2003 SARS epidemic – and found that it was very similar to SARS-CoV-2 despite the fact that SARS-CoV-2 has much more capacity to spread widely than SARS-CoV-1. The authors conclude that the "differences in the epidemiologic characteristics of these viruses probably arise from other factors, including high viral loads in the upper respiratory tract and the potential for persons infected with SARS-CoV-2 to shed and transmit the virus while asymptomatic." [24]

25. It is also possible that Covid-19 is transmitted as an aerosol – in other words, through the airborne route, *i.e.*, direct inhalation of virus suspended in the air. The study cited above also assessed the stability of aerosolized SARS-CoV-2 over time. To do this, they used a nebulizer to generate aerosols that would be similar to those observed in samples obtained from the upper and lower respiratory tract in humans. SARS-CoV-2 remained viable in aerosols throughout the duration of the three-hour experiment, suggesting that aerosol spread of SARS-CoV-2 is indeed possible. These findings are consistent with case reports of Covid-19 patients who were infected in settings in which they did not have direct contact with others. In one case, 45 people were diagnosed with Covid-19 after attending a choir practice in Washington State in early March although they had no direct physical contact with each other [25]. The findings are also consistent with a report in the journal, *Nature*, where researchers found viral RNA in aerosols sampled in

February and March at two hospitals in Wuhan, China. The levels of airborne viral RNA in well-ventilated patient rooms were relatively low but there were higher levels in some of the patients' toilet areas, presumably aerosolized by the flushing mechanism.

26. High levels of viral RNA were also found in areas where medical workers remove their protective equipment and in locations near the hospitals where people tended to congregate. The authors concluded: "Our study and several other studies proved the existence of SARS-CoV-2 aerosols and implied that SARS-CoV-2 aerosol transmission might be a non-negligible route from infected carriers to someone nearby."

27. The transmissibility of any infectious agent depends on several things: the probability of an infection event given a contact between a susceptible person and an infectious person; the duration of infectiousness – or number of days that a person can transmit – and the number of contacts that an infectious person has per unit time. This means that the transmissibility can vary in different settings and will depend on things like crowding, which increases the number of contacts. Based on a summary of multiple studies, each infectious person with Covid-19 is expected to infect between 2 and 3 people on average [26]. But this term – "on average" – obscures the substantial variability observed in different people. Some people are much more infectious than others and other people do not transmit at all. Like many other respiratory infections, SARS-CoV-2 follows the 20/80 rule – meaning that most transmission is associated with 20 percent of the infectious people while the other 80 percent infect relatively few people. The factors that

lead to this kind of "super-spreading" are not clear and it is thus not possible to identify in advance those people who are likely to infect a large number of other people.

28. Control of SARS-CoV-2 spread is also made more difficult because people can transmit the infection even when they do not have symptoms of the disease. This can happen in two ways. Many people with SARS-CoV-2 infection have few if any symptoms – as more and more seroprevalence studies are being conducted to identify who has been infected, it is estimated that 50-60 percent of infected people never develop symptoms of the disease. Seroprevalence surveys are studies that look for the presence of antibodies to an infection in a blood sample; these are only present in people who have been exposed to the infection and have mounted an immune response. Secondly, people who develop Covid-19 disease experience a "pre-symptomatic" period during which they are infected but do not yet have symptoms. A recent study in the *New England Journal of Medicine* found that quantitative SARS-CoV-2 viral loads were similarly high in four different symptom groups; people with typical symptoms of Covid-19, people with atypical symptoms, people who were pre-symptomatic, and those who remained asymptomatic [27]. Notably, 71 percent of the samples taken from pre-symptomatic persons had viable virus for one to six days before the development of symptoms. Because viral load is an accepted proxy for infectiousness, these data imply that a significant proportion of transmission events originate from persons who do not have detectable infection.

29. ***What kinds of interventions are currently available that could interrupt or reduce transmission of SARS-CoV-2?*** In the absence of a vaccine or pharmaceutical

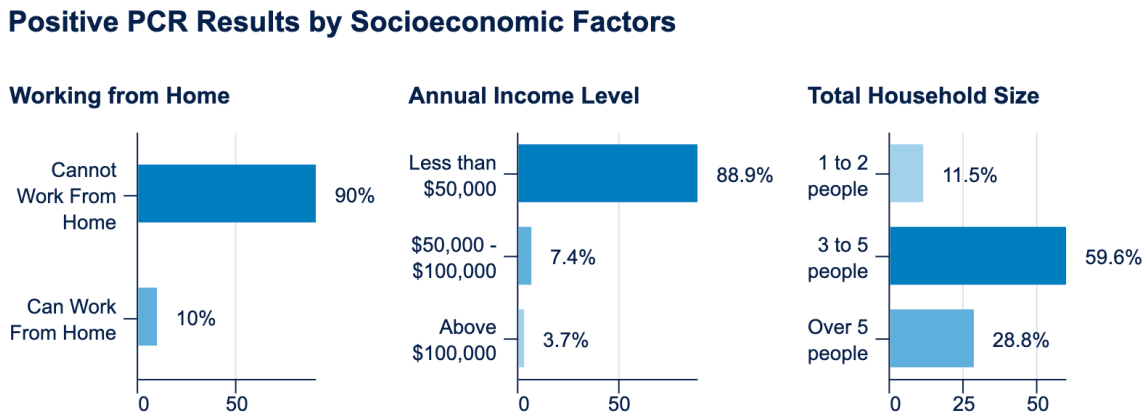
interventions that reduce the probability of transmission, there are a limited number of approaches to infection control, all of which involve restricting people's physical and social interactions. One can isolate people with symptomatic disease to try to prevent them from infecting others, but this will only be completely effective if people are diagnosed with the disease at or before the time that they become infectious. As noted above, in people who are infectious before they have symptoms or in infectious people who never develop symptoms at all, transmission can take place in the absence of symptoms. For diseases like this one, with significant asymptomatic spread, quarantine is used to separate and restrict the movements of people without signs of illness who may have been exposed to an infectious case so that they do not infect others during that period. Another approach is social distancing – this can range from asking people to stay at home or to avoid congregate settings such as schools, workplaces, or large gatherings. The purpose of social distancing is to reduce the number of person-to-person contacts one makes so that one is less likely to encounter an infectious person. Polls show that US adults practicing social distancing have 90% fewer contacts per day than those who are not social distancing. Those who completely or mostly isolate themselves generate about five contacts per day, compared with an average of 52 for those not attempting to isolate themselves [28].

30. It is challenging to directly measure the actual efficacy of the non-pharmacological interventions to reduce the spread of an infection because these interventions are not randomly assigned to individuals and then evaluated in a head-to-head comparison of what happens to people in intervention and non-intervention groups.

One approach to estimating the impact of social distancing measures is to conduct studies that screen entire communities to determine who is actively infected at the time of screening. The researchers then correlate various characteristics of the people screened with the likelihood that they have been infected. Few such studies have been conducted to date but one that is informative was conducted in the Mission District of San Francisco. The research team offered free Covid-19 testing to all persons ages four years and older in an area that includes approximately 5,700 people (29). Of nearly 3,000 residents and workers in a Mission District census tract who were tested in late April for active infection with the novel coronavirus, 62 individuals (2.1 percent) have tested positive.

Table 3 Viral test positivity in Mission District of San Francisco [29]

Figure 3. Number of ascertained coronavirus disease (cases over time calculated by mathematical model with adults reducing their contact by 25% (A, B); 75% (C, D); and 95% (E, F). We used parameter values of $R_0 = 2.26$, $\gamma = 1/5.02$, $\sigma = 1/5.16$. Dotted lines represent the beginning and end of the 6-week social distancing interventions, after which contact rates return to normal. For panels A, C, and E, intervention starts at day 50 after identification of first case; for panels B, D, and F, intervention starts at day 80 after identification of first case.



31. The question of the efficacy of quarantine, isolation and social distancing depends on when in the course of the infection most transmission is taking place. If most transmission occurs during the asymptomatic period – as it does, say, for HIV – isolation

of patients with disease will have little impact. If on the other hand, most transmission takes place when people have identified themselves as ill (as it did for SARS-CoV-1 in 2002), isolation can be a very effective way to reduce spread. The benefits of quarantine – restricting the movements of people who are known to be in contact with an infectious case – depend on how effectively one can identify all contacts and prevent them from mixing with the general public. For obvious reasons, this can be very challenging and can have unintended consequences if quarantined people are housed together and become infected in that setting. Social distancing cannot prevent all transmission but could have a substantial impact on delaying transmission since contact rates are often much higher in congregate settings such as schools, prisons and other residential facilities. None of these measures is likely to lead to complete control of an epidemic since transmission is expected to resume once these are discontinued. But they may delay spread and give health systems time to develop better responses to the disease, whether those are new drugs, vaccines or simply improved efficiency of supportive care.

32. ***I was asked to address the likelihood that voting at polling stations could lead to SARS-CoV-2 transmission and Covid-19 disease.*** Because voting takes place in public buildings where people congregate and because the risks of infection and disease in the North Carolina population are high due to the high prevalence of comorbidities, voting at a polling station in November entails a substantial risk of infection with Covid-19 that could result in symptomatic disease, hospitalization or death. The risk of an individual being infected during a community event in a public place depends on the number of

infectious people in that community at any particular time point and the number of physical, fomite-mediated and near contacts one makes during that process. To the extent that polling places are crowded, require people to wait in lines, involve interacting with polling staff or other voters at a close distance, move people through the process slowly, are poorly ventilated and/or involve people touching objects like pens, paper, or surfaces within the voting booth, they constitute a risk to voters. Similarly, if voters or poll workers use toilets that are also used by others, they can be put at risk. The data supporting some aerosol transmission of Covid-19 provides evidence that poorly ventilated areas where crowding may take place pose risk to those using these facilities. The probability that a person who is exposed to Covid-19 in this setting will go on to develop severe Covid-19 disease or to die depends on the age of that person and his/her underlying health status. Given the relatively high prevalence of relevant co-morbidities (obesity, hypertension, diabetes), the proportion of the population over 60, and the fact that older people are more likely to vote than younger people (on a nationwide basis, 66 percent of over 65 years compared to 35.6 percent of 18-29 years in the 2018 national mid-term elections [54]), there is a substantial risk that an infection with Covid-19 in North Carolina could result in symptomatic disease, hospitalization or death.

33. ***I was asked to address the likelihood of a persistent or increased risk of transmission of Covid-19 in the fall in the weeks/months leading up to November 3, 2020.*** Epidemiologists have projected a number of future Covid-19 epidemic trajectories based on a range of different possible scenarios but all of these scenarios are similar in that they

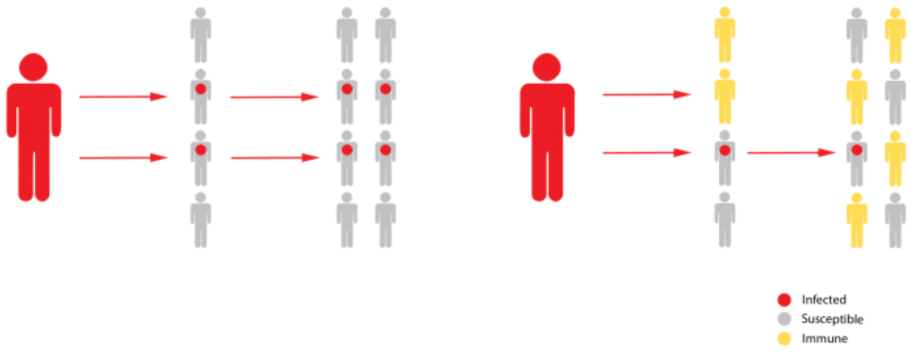
predict that it is highly likely that Covid-19 will continue to circulate at its current level or at an even higher level than currently in October and November of 2020. The likelihood of continued transmission of Covid-19 in the fall 2020 can be estimated by modeling the epidemic process. Mathematical models simulate epidemics under a variety of scenarios using "parameters" obtained from empirical (data-driven) studies. Typically, a model uses estimates of the relative transmissibility of an infectious agent, the average number of contacts people in different age groups make and the duration of infectiousness of the virus to reproduce the epidemic trajectory that has been observed. Then modelers introduce assumptions about the impact of interventions, for example, the number of social contacts that occur when social distancing measures are in place and re-run the model with these hypothetical parameters to determine what effect these changes will have. Over the past several months, multiple modeling teams have developed these kinds of models, and while they often obtain different results depending on various differences in the assumptions made, all show that reducing the number of social contacts, especially in the presence of asymptomatic infection, will "flatten" or reduce the epidemic curve. For example, one such model, reported in *Emerging Infectious Diseases* this week, investigated the effectiveness of social distancing interventions in a mid-sized city. Modeled interventions included reducing the number of contacts made by adults greater than 60 years of age, adults 20–59 years of age, and children under 19 years of age for six weeks. The modelers found that these interventions delay or flatten the epidemic curve and that even modest reductions of

contacts could reduce the number of new cases and deaths by 20 percent. Notably, however, when interventions ended, the epidemic rebounded [53].

34. The expected future trajectory of Covid-19 depends on a number of factors including the level of "herd immunity" that has already been achieved by the circulating of the infection, the extent of social mixing that occurs, and the possibility that SARS-CoV-2 will be more transmissible in cooler, drier weather.

35. First, herd immunity is achieved when enough people in a population have been infected and developed immunity so that the likelihood that an infectious person will come into contact with a susceptible person is low. This concept is illustrated in the graphic below. When an infectious person encounters only susceptible people, he or she can infect all of them but when most of the people an infectious person encounters are immune, relatively few people will be infected by that infectious case.

Table 4 Herd Immunity (From https://www.technologyreview.com/2020/03/17/905244/what-is-herd-immunity-and-can-it-stop-the-coronavirus/)



In a simple model of an outbreak, each case infects two more, creating an exponential increase in disease. But once half the population is immune, an outbreak no longer grows in size.

36. A general rule of thumb is that herd immunity can only be achieved when the proportion of people in a population who are immune is equal to $1-(1/R_0)$, where R_0

refers to the basic reproductive number of an infectious disease. This term is defined as the number of people who, on average, will be infected by a single infectious person in an entirely susceptible population. The basic reproductive number of SARS-CoV-2 is estimated between two and three, with an average of about 2.6. This means that about 60 percent of the population would need to be immune before we see Covid-19 cases level out (in the absence of interventions such as social distancing). At present, it is unclear what proportion of the US population is seropositive (in other words, has evidence of an immune response to the infection), but no study conducted in the US to date has suggested that more than 20-30 percent of any specific community is immune and most studies suggest that the number is closer to 2-3 percent. A recent study from Spain, one of the countries that has been most affected by the epidemic, found that only 2 percent of the population was immune [30]. Therefore, it is highly unlikely that, short of a catastrophic increase in circulating virus, herd immunity will be achieved by November 2020. Furthermore, the lack of herd immunity is in part due to social distancing that has taken place to date and this means that as a population, we remain highly vulnerable to epidemic spread.

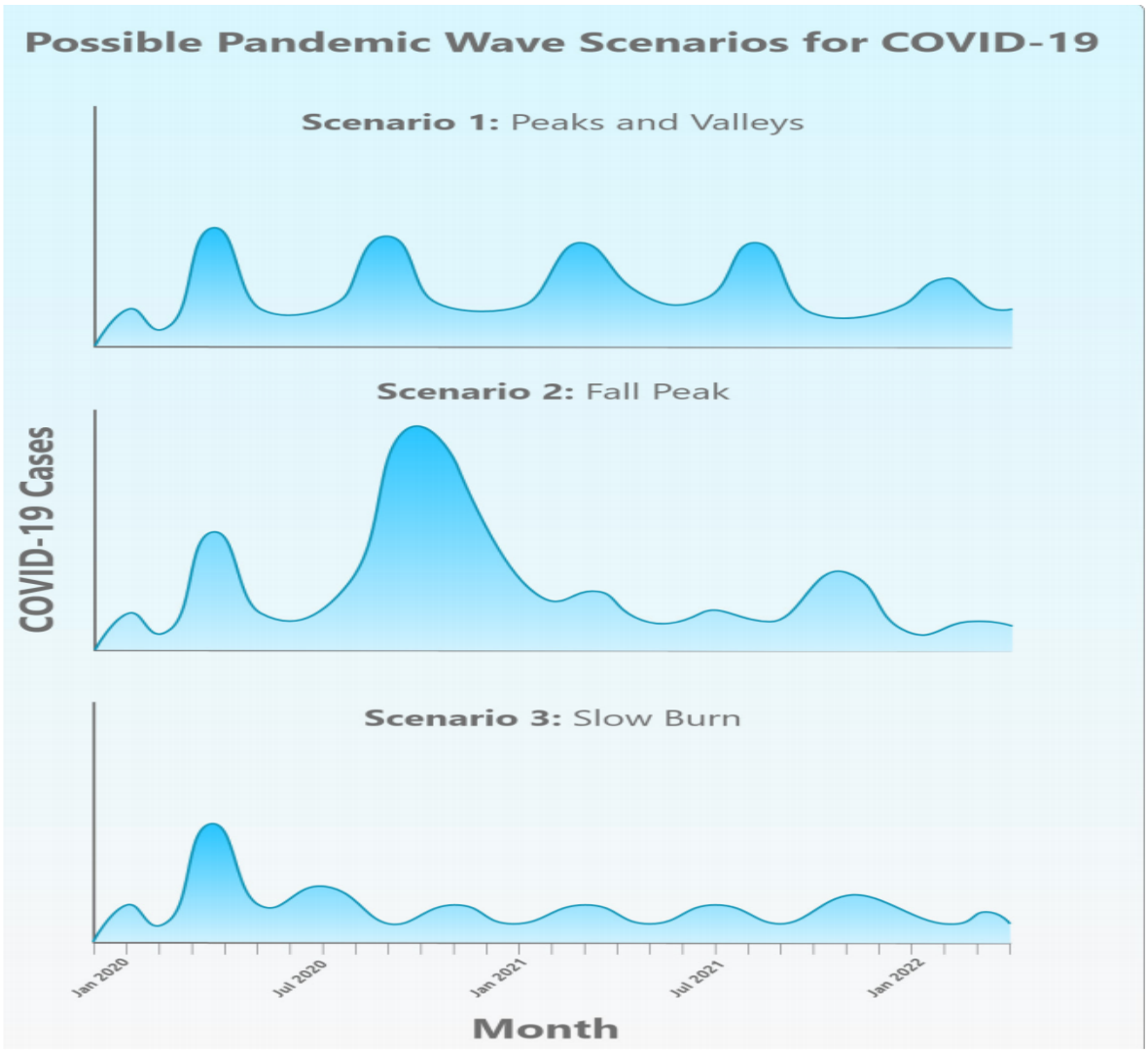
37. Secondly, it is highly likely that with the relaxation of social distancing measures and the end of "lock-down" the number of social contacts that people make will increase and that, therefore, the incidence of infection will increase accordingly. There is a linear relationship between the average number of social contacts individuals make and the reproductive number of the infection; as social contacts increase, the incidence of infection will increase proportionately.

38. Third, epidemic spread in the fall and winter could be driven by potential worsening of the epidemic due to changes in temperature or humidity that may be associated with higher viral stability with cooler and drier conditions, seasonal changes in host immunity and/or changes in human behavior (e.g., spending more time indoors). In the fall and winter, the outdoor air is colder, and the air is drier both indoors and out. For influenza, laboratory experiments have shown that absolute humidity — the amount of water vapor in the air — strongly affects viral transmission, with drier conditions being more favorable [31]. Lab studies on SAR-CoV-1 have also confirmed that viruses are stable for longer periods in cooler, drier environments [32]. However, multiple recent studies have suggested that SARS-CoV-2 transmission is possible in many different climates [33, 34].

39. Seasonal differences in transmission are also affected by differences in the ways people congregate in different seasons. In the fall and winter, people tend to spend more time indoors with less ventilation and less personal space than they do in the summer. Schools have been identified as the sites of much transmission of respiratory viruses including those that cause measles, chicken pox and influenza. [35, 36]. However, to date, the role of children in the transmission of SARS-CoV-2 is not clear and the relevance of the timing of school openings is not known. Finally, it is likely that host immunity is affected by seasonal changes. One hypothesis has focused on melatonin which has some immune effects and is modulated by the photoperiod [37], which varies seasonally. Vitamin D levels have also been associated with improved human immune responses -

these levels depend in part on ultraviolet light exposure which is higher in summer. There is strong evidence for the possible role of vitamin D supplementation in reducing the incidence of acute respiratory infection, as documented in a meta-analysis of randomized trials [38]. To summarize the evidence for seasonal trends in SARS-CoV-2, it is reasonable to expect that, like other beta-coronaviruses (a family of viruses with shared genetic characteristics), it may transmit somewhat more efficiently in fall and winter than summer.

Table 5 Possible Covid-19 scenarios [38]



40. These considerations have guided most of the modeling projections on the expected future trajectory of SARS-CoV-2 spread. The Center for Infectious Disease Research and Policy (CIDRAP) recently published a document where they drew lessons from previous influenza pandemics to predict the future trajectory of Covid-19 [39]. They summarize three different possible scenarios as illustrated in the above figure. In the first scenario, the first spring wave of Covid-19 that is currently underway will be followed by a series of repetitive smaller waves that occur through the summer and then consistently over a one- to two-year period, gradually diminishing sometime in 2021. These waves would be expected to vary geographically depending on what interventions are in place and how and when they are relaxed. Depending on the height of the peaks, this could lead to periodic re-implementation and interruption of social distancing measures over the next one to two years.

41. In the second and most likely scenario, the current first wave of Covid-19 will be followed by a larger wave in the fall or winter of 2020 and one or more smaller subsequent waves in 2021.

42. This pattern is what was seen with the 1918-19 influenza pandemic in which a small wave began in March 1918 but transmission leveled off during the summer months. This was followed by a much larger peak which occurred in the fall of 1918 and a third peak which occurred during the winter and spring of 1919. The 1957-58 and 2009-2010 influenza pandemics followed a similar pattern, with a smaller spring wave followed by a much larger fall wave [40]. Given the many similarities between how SARS-CoV-2 and

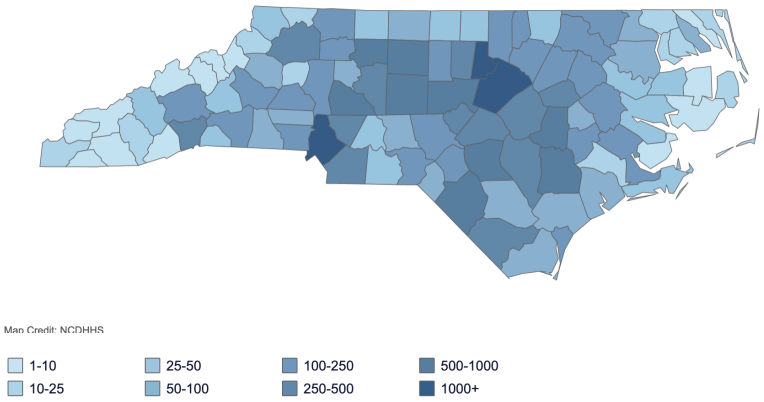
influenza are spread, it is expected that Covid-19 will behave in a similar way, and most epidemiologists expect that incidence will increase in the fall and winter months of 2020-2021.

43. In the third scenario proposed by the CIDRAP team, the first wave of Covid-19 in spring 2020 would be followed by persistent ongoing transmission and disease incidence without a clear wave pattern. This third scenario might not lead to the reinstitution of mitigation measures, although cases and deaths will continue to occur especially in areas where risk factors for disease and death are common.

44. Whichever scenario the pandemic follows, it is highly likely that Covid-19 activity will continue for at least another 18 to 24 months, with hot spots arising periodically in diverse geographic areas. In the period prior to the widespread use of an effective vaccine, this spread will continue to lead to serious disease and death in at-risk groups As the pandemic wanes, it is likely that SARS-CoV-2 will continue to circulate at lower levels in the human population and will synchronize to a seasonal pattern with diminished severity over time, as other coronaviruses, such as the beta-coronaviruses OC43 and HKU1, [41] and past pandemic influenza viruses have done.

45. ***I was asked to describe the Covid-19 situation in North Carolina.***

Table 6 County-specific Covid-19 prevalence for North Carolina by May 21, 2020. (from https://covid19.ncdhhs.gov/)

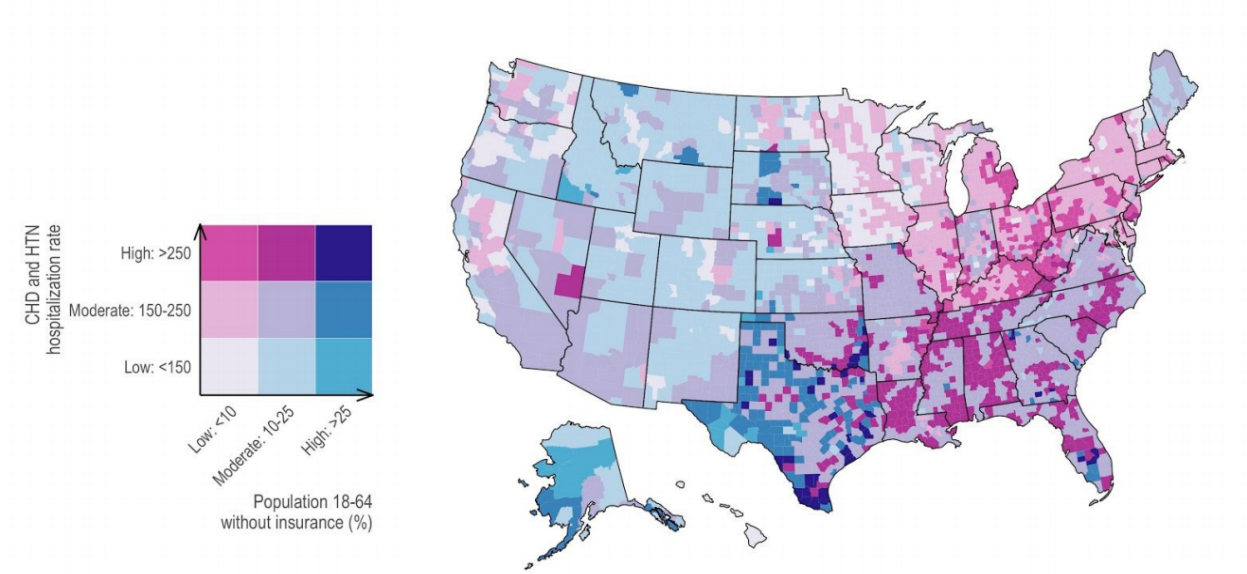


46. To assess the risk of serious disease given a Covid-19 infection, we can turn to the existing data on the prevalence of specific risk factors in the state. The CDC has documented that 33 percent of North Carolinians are obese and an additional 35 percent are overweight; 35 percent have a diagnosis of high blood pressure and 13.1 percent have diagnosed diabetes mellitus [45, 46, 47]. 16.3 percent of the state's population is 65 years old or over [48]. It is useful to compare the prevalence of different co-morbidities associated with poor outcomes in North Carolina to other states. The figures below give county-level rates of hospitalizations for coronary heart disease and hypertension demonstrating that North Carolina has comparatively high rates of these diseases and the age distribution of the population**.**

47. It is useful to compare the prevalence of different co-morbidities associated with poor outcomes in North Carolina to other states. The figures below give county-level rates of hospitalizations for coronary heart disease and hypertension demonstrating that

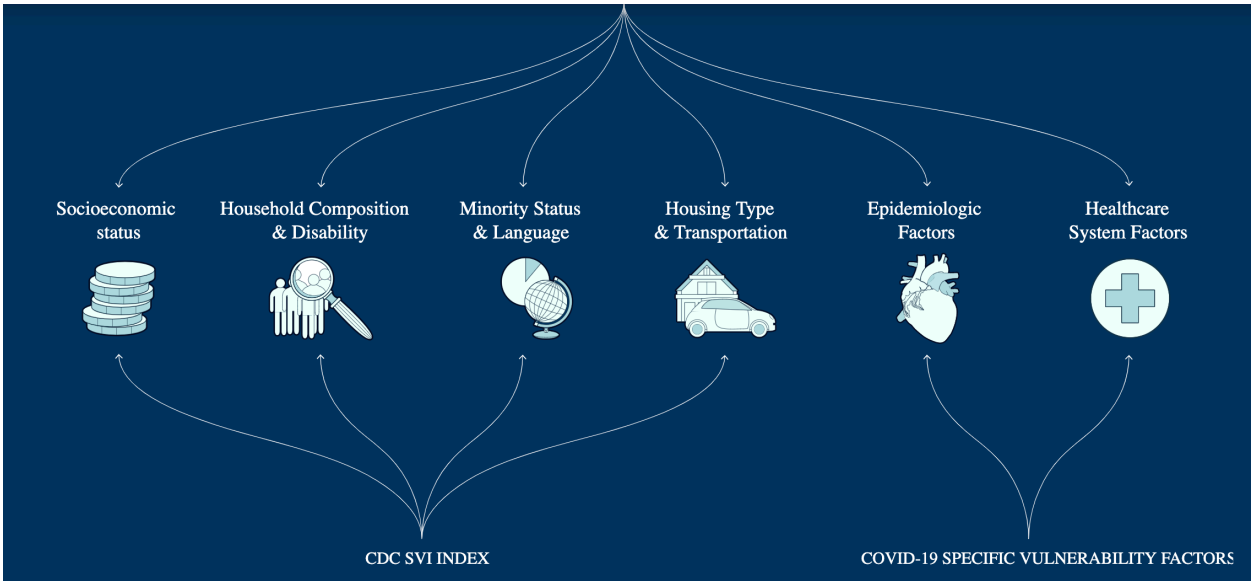
North Carolina has comparatively high rates of these diseases and the age distribution of the population**.**

Table 7. Rates of hypertension and Coronary Heart Disease Nationally. From (https://www.medrxiv.org/content/10.1101/2020.04.08.20058248v1)



48. The Kaiser Family Foundation has developed a method to estimate the proportion of a state's population at elevated risk for serious Covid-19 illness [49]. Using data from the CDC's 2018 Behavioral Risk Factor Surveillance System (BRFSS), they estimated the total number of at-risk adults by state—based on the revised definition from the CDC—of adults who are at higher risk of serious illness if they get infected with coronavirus. The relevant factors include ages 65 or older, heart disease, chronic obstructive pulmonary disease (COPD), uncontrolled asthma, diabetes, or a BMI greater than 40. Based on this analysis, 39% of adults over age 18 in North Carolina are at risk for serious disease with older adults making up 54.2% of those at high risk.

Table 8 CCVI Score components [48]



49. In another nation-wide assessment of risk, the Surgo Foundation has developed a Covid-19 community vulnerability index (the CCVI) to identify communities at especially high risk of being affected by Covid-19 [50]. The CCVI combines indicators specific to Covid-19 with the CDC's social vulnerability index, which measures the expected negative impact of any type of disaster. The indicators are based on the themes listed below.

50. On this scale, North Carolina scored a 98 out of 100 with 100 being the most vulnerable. It ranked second among the states on this measure, mostly because its high (poor) score in the area of healthcare system factors. These data suggest that in the event of further spread of Covid-19, North Carolina may experience higher levels of disease, disability and death than other states experiencing the same amount of transmission.

51. ***I was asked if herd immunity, progress in vaccine development or the development of drugs to treat Covid-19 will alter the expected course of the Covid-19***

pandemic in the United States and specifically North Carolina. To date, the proportion of the population that is likely to be immune is far less than that that required to achieve herd immunity. This is unlikely to change significantly before November. Although recent studies of the temporal trajectories of the appearance of SARS-CoV-2 antibodies show that most people who are infected with the virus do develop an antibody-mediated immune response, it is not yet clear whether this response is adequate to protect people from future infection or for how long it might be protective. Other coronaviruses, such as those that cause colds, are known to provide protection for periods of approximately one year and this experience has led most Covid-19 experts to accept the "educated guess" that after being infected with SARS-CoV-2, most individuals will have an immune response which will offer some protection over the medium term — at least a year — and then its effectiveness might decline. Until there is empirical evidence of how well-protected previously infected people are in the future, there is no way to confirm or deny the existence of long-term immunity.

52. An effective vaccine is extremely unlikely to have been developed, tested and widely distributed before November. Vaccine development has proceeded at an unprecedented pace. More than 110 candidate vaccines are under development. A number of companies and research teams already have candidate vaccines that are either in human trials (eight have started) or close to ready to trial in humans. The most advanced of these seems to be the ChAdOx1 nCoV-19 vaccine being developed by a group in Oxford, England. The speed with which these vaccines are being developed is partly due to the fact

that a great deal of work was done on a SARS-CoV-1 vaccine after the 2002 epidemic and some of that work can be applied to this organism.

53. Despite this extraordinarily rapid progress, it is important to realize that the usual time frame from development to widespread use of a vaccine is over ten years. New vaccines require a complex set of trials to establish safety, immunogenicity, optimal dosing, etc. Phase 1 trials are usually conducted in small groups of healthy volunteers and are designed to establish whether serious adverse effects occur with escalating doses of the agent and whether the vaccine produces the expected immune response. Phase 2 trials are designed to replicate Phase 1 results in a more diverse population of volunteers, to assess whether the expected immune response is generated, and to test different vaccine schedules. Once safety, immunogenicity and optimal dosing are established, Phase 3 studies are conducted to determine vaccine efficacy. Phase 3 studies are usually much larger than phase 1 or 2 studies and are conducted in people at risk for the infection in question. So the time frame of these trials depends on the actual incidence of infection and is expected to be shorter in regions with very high rates of disease. The completion of all three steps is required for a vaccine to be approved by the FDA. Once a vaccine is approved, it must then be manufactured at a scale that will provide adequate coverage for a large population.

54. The White House has recently announced an initiative, "Operation Warp Speed," to expedite the development of a vaccine that will be available to the US population. Although many scientists question the timeline proposed by the project, the

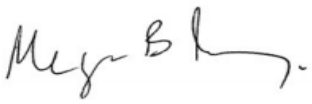
goal is to speed up the development and production of a vaccine so that 100 million doses are available in November of 2020 and the remaining 200 million doses needed to vaccinate the US population are ready by early 2021. Thus, even in the most optimistic scenario, it is highly unlikely that a vaccine will have been distributed and had time to induce an immune response in a significant number of Americans by November 3, 2020.

55. Although new and repurposed drugs are being tested and some may be found to be helpful in treating severe Covid-19, this is unlikely to have a major impact on the transmission of the virus and the risk of severe disease or death by November 2020. A number of antiviral drugs are currently being developed and other existing drugs are being "repurposed" as potential therapies for Covid-19. The hope is that these drugs will reduce the rate of death and severe disease in people who are treated with them. As of mid-April, the FDA website had listed 72 active and 211 planned Covid-19 drug trials and almost 1000 drug-development proposals have been submitted to the agency. To date, only Remdesivir has been shown in a major, randomized control trial to reduce the duration of illness in Covid-19 patients. In that study, Remdesivir reduced the median time to recovery in hospitalized patients with advanced Covid-19 disease and lung involvement from 15 days for those who received placebo to 11 days for patients treated with Remdesivir [51]. The researchers also noted a survival benefit (which was not "statistically significant") with the Remdesivir group experiencing an 8.0 percent mortality rate compared to 11.6 percent for the placebo group. This suggests that even with the approval of the drugs that have been

found to be effective in clinical trials, people with severe Covid-19 are at risk for death as well as the long-term effects of lung damage and other sequelae of infection detailed above.

I declare under penalty of perjury that the foregoing is true and correct.

Executed this 2nd day of June, 2020.



Megan Murray, MD, MPH, ScD

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2017-	Ronda Stryker and William Johnston Professor of Global Health	Global Health and Social Medicine	Harvard Medical School

Appointments at Hospitals/Affiliated Institutions

01/98-09/02	Clinical Assistant in Medicine	Dept. of Internal Medicine	Massachusetts General Hospital
09/02-09/06	Assistant in Medicine	Dept. of Internal Medicine	Massachusetts General Hospital

09/06-02/08	Assistant Physician	Dept. of Internal Medicine	Massachusetts General Hospital
02/08-09/10	Consultant (Medicine Services)	Dept. of Internal Medicine	Massachusetts General Hospital

Other Professional Positions

Year	Position Title	Institution	Level of effort
1980-1984	Refugee Camp Coordinator	Intergovernmental Committee for Migration, Phanat Nikhom, Thailand	
1984	Public Health Educator	Matanyok Rural Training Project, Rift Valley, Kenya	
2004-	Research Director	Partners In Health, Boston	36 days per year

Major Administrative Leadership Positions

Local

2007-	Director of Research	Division of Global Health Equity, Brigham and Women's Hospital and Partners In Health, Boston, MA
2010-	Director	Research Core, Department of Global Health and Social Medicine, Harvard Medical School, Boston, MA
2012-2013	Member	Executive Leadership Team, Global Health Delivery Partnership, Harvard Medical School, Brigham and Women's Hospital and Partners In Health, Boston, MA

Committee Service

Local

1986-1990	Curriculum Committee	Harvard Medical School Member
2001-	Infectious Disease-Epidemiology Graduate Admissions Committee	Harvard School of Public Health Member
2003-2005	Faculty Council	Harvard School of Public Health Member
2004-	Steering Committee for the Residency in Global Health	Brigham and Women's Hospital Member
2005-	Human Subjects Committee	Harvard School of Public Health Member
2005	Task Force on Women in Sciences and Engineering	Harvard University, Cambridge, MA Member

2006	President's Task Force on Avian Influenza	Harvard University Member
2006	Search Committee for Dean of Educational Programs	Harvard School of Public Health Member
2007	Epidemiology Curriculum Committee	Harvard School of Public Health Member
2007	Search Committee for Compliance Officer	Harvard School of Public Health Member
2007	Search Committee for Assistant Professor in the Division of Social Medicine and Health Inequalities	Brigham and Women's Hospital Member
2008	Search Committee for Assistant Professor in Infectious Disease Epidemiology	Harvard School of Public Health Member
2009	Search Committee for Assistant Professor in Infectious Disease Epidemiology	Harvard School of Public Health Chair
2010	Global Health Epidemiology Committee	Harvard School of Public Health Member
2010-2011	Strategic Leadership Team	Brigham and Women's Hospital Member (Co-Chair Community Engagement Mission Area)
2012	Ad Hoc Committee to Evaluate Professorial Candidate	Harvard Medical School Member
2012-2016	Professor of Population Medicine Search Committee	Harvard Pilgrim Health Care Institute Member
2012	Ad Hoc Evaluation of Professorial Appointment Committee	Harvard Medical School Member
2012	Global Health Instructor Search Committee	Harvard Medical School Co-chair
2014-2016	Pershing Square Professorship in Global Health Search Committee	Harvard Medical School Chair
2015	Search Committee for Professor of Biomedical Informatics	Harvard Medical School Member

2016-	Department of Biomedical Informatics Executive Committee	Harvard Medical School Member
2016-	Committee on Promotions, Reappointments, and Appointments (P&R)	Harvard Medical School Member
2018-2019	Dean's Innovation Grants Review Committee	Harvard Medical School Member
2018-2019	Therapeutics Planning Foundry Committee	Harvard Medical School Member
2019-	Center for Computational Biomedicine (CCB) Advisory Committee	Harvard Medical School Member
2019-	Faculty Council	Harvard Medical School/Harvard School of Dental Medicine Member
2020-	Ariadne Spark Grant Review Committee	Harvard Medical School Member
2020-	Massachusetts Consortium on Pathogen Readiness (MassCPR) Working Group on Epidemiology	Harvard Medical School Co-Lead

National and International

2005-2007	Committee on Infectious Diseases among Gulf War Veterans	Institute of Medicine, Washington, DC Member
2006-2010	IHR Roster of Experts in Modeling Analytical Epidemiology	World Health Organization, Geneva, Switzerland
2007-2009	Global Task on XDR Tuberculosis	World Health Organization
2007	External Review Committee for TB Program	Montreal Chest Institute, McGill University Montreal, Canada Member
2008-2011	STAG (Strategic and technical advisory group) TB	World Health Organization Member

2008	39th Union World Conference 2008 Drug Resistance /MDR-TB management II	International Union TB and Lung Disease Paris, France, Coordinator
2009	Panel to Review the DST/NRF Centre of Excellence for Biomedical TB Research	National Research Foundation, Pretoria, South Africa, Convener
2009-2010	Expert Panel on Tuberculosis and Diabetes	Union of TB and Lung Disease and World Diabetes Federation Member
2010-2015	Advisory Group to Fogarty Grant	Member Public Health Research Institute New Jersey
2010-	Working Group on New Diagnostics	Member, Stop TB Partnership Geneva, Switzerland
2013-2014	Millennium Villages Project Independent Expert Group Meetings	Member Earth Institute, Columbia University Millennium Development Goals Centre West and Central Africa Dakar, Senegal
2013-2014	External Advisory Committee on Tuberculosis	Member Gates Foundation New York City, NY
2016-	Critical Path to TB Drug Regimens (CPTR) Initiative	Member

Professional Societies

1995-	Infectious Disease Society of America	Member
1997-	Society for Epidemiologic Research	Member
2007-	International Union of TB and Lung Disease	Member
2007-	Global Health Council	Member
2009-	American Society for Tropical Medicine	Member

Grant Review Activities

2004	Fogarty International Center/NIH Study Section ZRG1 ICP-3(03)	NIH, Bethesda, MD Member
2005	Improving Tuberculosis Control in Africa; Mathematical Modeling of Intervention Trials	Wellcome Trust Review London, England Member
2008	Postdoctoral Program Review for Indonesian PhDs	Royal Netherlands Academy of Arts and Sciences (KNAW), Amsterdam, Holland Referee
2008	Center for AIDS Research Scholar and Feasibility Scientific Reviewer Committee	Harvard School of Public Health Member
2009, 2010	Center for Scientific Review/NIH study section l ZRG1 IDM-P 50 R	NIH Ad hoc Member
2009-2016	Center for Scientific Review/NIH Study Section on Clinical Research and Field Studies of Infectious Diseases (CRFS)	NIH Permanent Member
2009	Wellcome Trust London, England	Ad hoc Reviewer
2010	National Science Foundation South Africa	Ad hoc Reviewer
2012	Center for Scientific Review/NIH Study Section ZRG1 AARR-K (52)	NIH Ad-hoc Member
2015	Center for Scientific Review/NIH Study Section ZRG1 IMST-K (50)S	NIH Ad-hoc Member
2017	Center for Scientific Review/NIH Study Section ZAI1 LG-M (M1) NIAID Clinical Trial Implementation Cooperative Agreement (U01/R01)	NIH Ad-hoc Member
2018	Center for Scientific Review/NIH Clinical Research and Field Studies of Infectious Diseases [CRFS] Study Section ZRG1 IDM-R (02)	NIH Ad-hoc Member

2019	Center for Scientific Review/NIH Study Section ZRG1 IDM-R (50)	NIH Ad-hoc Member

Editorial Activities

Ad hoc Reviewer

Science
Nature Medicine
New England Journal Medicine
Lancet
Lancet Infectious Diseases
Lancet Pulmonary Medicine
Epidemiology
American Journal of Epidemiology
International Journal of Tuberculosis and Lung Diseases
British Medical Journal
British Medical Journal, Global Health
Emerging Infectious Diseases
PLoS Medicine
PLoS Pathogens
PLoS One
Scandinavian Journal of Infectious Disease
Journal of the American Medical Association
American Journal Respiratory and Critical Care Medicine
Journal of Clinical Microbiology
Antimicrobial Agents and Chemotherapy
PNAS (Proceedings of the National Academy of Sciences)
Bulletin of the World Health Organization
Clinical Infectious Disease
Journal Infectious Disease
Annals of Internal Medicine
American Journal of Tropical Medicine and Hygiene
Royal Society Proceedings B
BMC Medicine
BMC Genomics
BMC Public Health
BMC Biology
BMC Health Services
Interface
Epidemics
Royal Society Open Science

mBio

Other Editorial Roles

2004-	Member of Editorial Board	European Journal of Epidemiology
2005-2012	Associate Editor	International Journal of TB and Lung Disease
2009-	Associate Editor	PLOS Medicine

Honors and Prizes

1980	Dartmouth General Fellowship	Dartmouth College
1990	Aesculupian Society	Harvard Medical School
1990	Paul Dudley White Fellowship	Harvard Medical School
1996	Howard Hughes Post-Doctoral Research Fellowship	Howard Hughes Medical Institute
1997	Tapplin Fellowship Award	Harvard School of Public Health
2001	Teaching Award	School of Public Health, Boston University
2002	Teaching Award	Harvard School of Public Health
2004	Ellison Senior Scholar	Ellison Medical Foundation
2008	Recognition Award	Harvard School of Public Health
2010	Nominated for Mentorship Award	Harvard Medical School
2010-11	Landolt Chair	Ecole Polytechnique Federale de Lausanne

Report of Funded and Unfunded Projects

Funding Information

Past

1997-2003 Molecular epidemiology of tuberculosis
NIH/NIAID 1K08AI001430-01
PI
This study explored the use of molecular epidemiologic data for epidemiologic inference and evolutionary studies of *M. tuberculosis*.

2000-2003 Population-based investigations of tuberculosis
NIH R01AI046669
Co-investigator
In this project, population-based genetic studies of human specimens were used to determine the clinical consequences of mutations in genes associated with bacterial antibiotic resistance and virulence.

2002-2005 Transmissibility and fitness of drug-resistant TB, Sverdlovsk
WHO T9-181-270
PI
This project assessed epidemiologic risk factors for TB drug resistance, identified locally prevalent drug-resistance profiles, used molecular epidemiological analyses to measure association between clustering and specific drug-resistance mutations, and assessed demographic distribution of drug resistance in prison and local community, evaluating extent of transmission between the two groups.

2003-2005 INH resistance in Beijing/W Isolates of *M. tuberculosis*
NIH R21 AI055800
Co-investigator
This project sought to identify risk factors associated with Isoniazid resistance which may be pathogen and/or host- specific and which may lead to acquisition of MDR-TB, after controlling for compliance.

2003-2005 Decision analysis for TB control
Bill Melinda Gates Foundation
Co-investigator
This project developed a decision-analytic model that could be used with data from different countries to assess the potential benefits, costs, and cost-effectiveness of the full range of policy options for dealing with MDR-TB, including preventive therapy, active case finding, diagnostic testing and treatment.

2004-2009 Curriculum in Emerging Infectious Diseases
NIH/NIGMS K073000-04
PI ($363,933)

This project aimed to develop and implement a core course in transmission dynamics of emerging infectious diseases, taking an interdisciplinary approach that incorporates case-based seminars and short courses.

2005-2007 Evaluation of a community based HIV-TB adherence support program in a government ARV-rollout site in KwaZulu-Natal, South Africa
Harvard University Center for AIDS Research
Co-investigator
This study evaluated the feasibility of community-based adherence support program designed to improve HIV/AIDS and TB outcomes among a cohort of HIV patients in a government ARV treatment program.

2006-2007 Ferroportin Polymorphisms and Tuberculosis Susceptibility
William F. Milton Fund/Harvard Medical School
PI
This study assessed the association between Ferroportin (FPN1) mutations, iron intake and TB susceptibility in South Africa.

2006-2010 Macrophage Iron Metabolism and Tuberculosis Infection
NIH/NIAID R21 AI068077-01
PI ($271,375)
We elucidated the role of host macrophage iron status on the growth of *M. tuberculosis* and explored the impact of iron and ferroportin status on cellular immune function.

2006-2011 Epidemiology of Multidrug-Resistant Tuberculosis in Peru
NIH/NIAID R01 A1057786-01A2
Co-investigator
The goal of this project is to provide new knowledge about the transmission dynamics of multidrug-resistant tuberculosis in a high TB-burden area in Peru and will measure within-household transmission of various strains of TB, assess the impact of socio-demographic and clinical confounders and risk modifiers, and measure associations between specific resistance mutations and phenotypes.

2007-2009 A Postmortem Study of the Burden of MDR and XDR Tuberculosis Among Adult Inpatient in KZN Deaths Occurring at Edendale Hospital Kwazulu-Natal South Africa
Massachusetts General Hospital
PI ($60,050)
This study estimated the burden of tuberculosis among seriously ill individuals in KZN and measured the proportion of TB among these patients which is drug-resistant by conducting postmortem tests at Edendale Hospital KZN.

2007-2014 Epidemiology and Transmission Dynamics of MDR/XDR Tuberculosis
NIH/NIAID U19 A1076217
PI ($13,422,751)
We conducted a series of linked interdisciplinary research projects focused on the emergence and transmission of multidrug and extensively drug resistant TB: a cohort

study of host and microbial factors associated with MDR and XDR TB in Lima, Peru; a study characterizing *M. tuberculosis* strain diversity and its contribution to the emergence and spread of MDR; and a study using epidemic and individual predictive models to support public health policy and clinical decision-making for MDR and XDR TB.

2009 Systematic Reviews of Diabetes and Tuberculosis Interactions
PI ($25,000)
International Union of TB and Lung Disease
We evaluated the links between TB and diabetes by conducting a series of systematic reviews and meta-analyses.

2009-2012 Bioaerosols Production and Influenza Study
Pulmatrix Inc.
PI ($348,393)
The project measured the particle production in persons diagnosed with active influenza, measured the quantity and size distribution of influenza virus particles generated and exhaled by persons infected with influenza during normal tidal breathing, and measured the secondary attack rate of influenza within their households.

2009-2013 Treat TB: Technology, Research, Education and Technical Assistance for TB
USAID (subcontract through International Union against TB and Lung Disease)
Co-investigator
The subproject aims were to develop a modeling tool to assist national policy-makers in selecting the appropriate tests and strategies for the diagnosis of tuberculosis in specific types of epidemiological settings, with an emphasis on low- and middle-income countries, taking into account a variety of modifying factors including drug resistance and HIV.

2009-2014 Strengthening and Studying Community Based Integrated Primary Health Care Systems in Rural Rwanda
Doris Duke Foundation
Co-investigator
The PHIT Partnership strengthened integrated primary health care delivery in Rwanda. The Partnership deployed a care-based intervention, conduct implementation research to generate data for ongoing monitoring, evaluation, and quality improvement of the intervention.

2009-2014 MIDAS Center for Communicable Disease Dynamics
NIH/NIGMS U54 GM088558-01
Co-investigator
This project advanced the quantitative study of communicable diseases through training/education, transdisciplinary research, and public health policy and will develop statistical and novel modeling methods, train mathematical modelers, perform outreach, and develop software for the analysis of communicable disease data.

2012-2013 Identification of GyrA/B Mutations that Predict Fluoroquinolone Resistant TB

Harvard University Center for AIDS Research
Co-investigator
This project evaluated the correlation between newly-developed molecular genetic probes that can detect mutations in the gryA and gryB genes of tuberculosis which may render them more resistant to first and later generation quinolones.

2013-2014 African Health Facility Capacity to Roll Out Technological Interventions
Gates Foundation
PI ($17,232)
This project summarized the following outcomes across Rwanda health facilities: the percent and number of health facilities with electricity currently; estimate percent of health clinics with electricity within five years; percent and number of facilities with rapid HIV testing available; and the distribution of HIV testing staffing.

Current

2014-2020 Integrated discovery and development of innovative TB Diagnostics
NIH/NIAID CETR U19AI109755
PI ($29,218,333)
This multi-disciplinary collaboration is designed to enable the discovery of new biomarkers of *Mycobacterium tuberculosis* drug resistance, identify optimal clinical sampling strategies directed toward detection of *Mtb* DNA and develop and test a sensitive micro-array based rapid diagnostic. Our long-term goal is to develop a diagnostic strategy that will improve the diagnosis of childhood and DR TB and stem the further spread of the disease. *This grant is in a no cost extension phase.*

2015-2022 Metabolic Factors that Control the Spectrum of Human Tuberculosis
NIH/NIAID TBRU U19AI111224
Co-PI ($19,815,180)
This consortium project focuses on the link between host immune and metabolic factors and their impact on progression and persistence of tuberculosis. Teams focusing on human subjects, bio-informatics, and metabolomics work in parallel to identify targets including pathways linking human metabolism and immune response, T cells involved in *Mtb* response, pathogen determinants of drug resistance and pathogen-shed markers of clinical TB phenotypes. Each project includes validation of these targets in the guinea pig model.

2018- Metabolic Factors that Control the Spectrum of Human Tuberculosis
NIH/NIAID TBRU U19AI111224-04 Supplement
Co-PI ($200,000)
This supplement to the TBRU consortium project is a new collaborative, multi-disciplinary effort that conducted a genome-to-genome approach aimed at the identification of

interacting molecular patterns in *Mtb* and the human host. The same approach and new methods will be adaptable and easily applicable to other populations being studied within the TBRU program.

2019-2024 Bacterial Determinants of Treatment Response in Mycobacteria Tuberculosis
NIH/NIAID U19AI142793-01
PI ($14,633,712)
This study will focus on the discovery of the genetic determinants of drug tolerance and resistance in mycobacteria tuberculosis both through mechanistic bench studies and through a genome wide association study of treatment failure in TB patients.

2019-2021 Randomised trial of an intervention to increase tuberculosis notifications by private practitioners in Indonesia, plus sequencing and susceptibility sub studies
CRDF Global u/d USDA (59-0210-06-004) DAA3-19-64909-2
United States Research Leader ($99,917)
This study will evaluate whether a tailored intervention package increases notifications of tuberculosis (TB) by private practitioners in Bandung, Indonesia.

2020-2023 Are TB neighbourhoods a high risk population for active intervention?
CRDF Global u/d NIAID
United States Research Leader ($99,999)
This study will confirm whether neighborhoods around known, routinely diagnosed TB index cases are high risk sub-populations which may warrant active intervention to enhance TB control.

Unfunded Projects

2003 Transmission dynamics of SARS (Co-leader)
I co-led a team that developed a mathematical model of the transmission dynamics of SARS. (Lipsitch et al. Science 2003)

2005-2011 TB Genome Project (Collaborator)
Whole genome sequencing of sets of drug resistant *M. tuberculosis* isolates.
I led a collaboration with the Broad Institute to identify, sequence and analyze progressively resistant isolates of *M. tuberculosis* to identify drug resistance mutations and to characterize compensatory or enabling mutations. We currently have one manuscript under review and several in preparation.

2006 Cost-effectiveness of testing the blood supply for West Nile Virus (Supervisor)
I supervised a doctoral student in the development of a combined transmission/cost-effectiveness model on West Nile Virus. (Korves et al. PLoS Med 2006; Korves et al. Clin Infect Dis 2006)

2006-2010 Determinants of tuberculosis (Advisor)
I supervised two doctoral students to carry out epidemiologic studies and meta-analyses of the associations between determinants (smoking and diabetes mellitus) and tuberculosis and to use the parameters thus obtained to construct mathematical models assessing the impact interventions directed at these determinants. (Jeon et al. PLoS Med 2008; Jeon et al, Trop Med and Int Health 2010; Lin et al. Lancet 2008; Lin et al. Am J Respir Crit Care Med 2009, Murray M et al, IJTLD 2010, Baker M et al. BMC Medicine 2011, Lin et al. IJTLD 2011). I supervised Dr. Olivia Oxlade on work that is a further extension of this project.

2007-2009 Timing of ART in patients co-infected with HIV and TB in Rwanda: an observational approach (Initiator)
I initiated this project and supervised a doctoral student in the collection and analysis of the data. This work led to a paper published in PLoS Medicine (Franke M et al. PLoS Med 2011).

2007-2009 Metabolic modeling of *M. tuberculosis* (Collaborator)
I collaborated with a team of bio-informaticists on a project to fit a metabolic flux model to *M. tuberculosis* expression data to mycolic acid production. (Colijn et al. PLoS Computational Biology, 2009)

2008-2009 Structural analysis of *M. tuberculosis* "resistome" (Collaborator)
I collaborated with George Church on a project to define the structural basis of drug resistance in *M. tuberculosis* using sequence data. We published one paper together (Sandgren et al. PLoS Med 2009).

2008-2010 *M. tuberculosis* isoniazid and quinolone mono-resistance in South Africa (Mentor)
I supervised two trainees who are investigating the frequency and outcomes of mono-resistance in *M. tuberculosis* in South Africa. We published two papers in this area. (Jeon C et al, 2010, Jacobson K et al, 2011).

2008-2010 ART Outcomes in Rwanda for 1000 HIV patients (Co-investigator)
I provided technical support and supervised the data collection and analysis team. We have published a paper on this topic (Rich et al, 2011).

2009-2010 Within host dynamics of TB and the evolution of drug resistance. (Initiator)
I collaborated with my former trainees, Ted Cohen and Caroline Colijn, on a project to model the within-host evolution of drug resistance (Colijn C et al, PLoS One, 2011).

2009-2011 Sex trafficking and HIV transmission in India (Advisor)
I supervised a doctoral student in the analysis of data and construction of a mathematical model of HIV transmission among trafficked sex workers in India. We published several papers together.

2010-2012 Cholera transmission in the Democratic Republic of the Congo and Haiti. (Collaborator and Adviser). I worked with a team including hydraulogists and infectious disease modelers on the transmission routes by which cholera spreads. We published three papers (Rinaldo et al, Proc Natl Acad Sci, 2012; Bompangue et al, PLoS Curr 2012; Bompangue et al, Lancet, 2012).

2012- Poverty traps in under-resourced settings (Collaborator)
I collaborate with Matthew Bonds on a range of studies to understand the role of infectious diseases in creating poverty traps in Rwanda and other under-resourced settings.

2014-2015 MDR TB in India
I worked with a Fulbright fellow to assess the burden of MDR TB in India.

2013-2016 Ebola Diagnostics, Asymptomatic Infection and Modeling (Initiator and Collaborator)
I worked with the Partners in Health clinical teams in Sierra Leone to evaluate two point of care diagnostic tests and supervised Gene Richardson in a study of asymptomatic Ebola infections and Ibrahim Diakite on a study of dynamic modeling of Ebola vaccination strategies.

2012- Impact of Health Research Capacity Building (Team Leader)
I lead a team focused on the implementation and assessment of Health Research Capacity Building in Africa.

2014- Yaws epidemiology and impact of mass drug administration (Collaborator)
I work with my former student, Eric Mooring, on the evaluation of data collected during a mass drug administration campaign in Papua, New Guinea.

2014- Health System Strengthening in Madagascar (Collaborator)
I work on developing methods to evaluate the impact of health system strengthening in Madagascar and other implementation sites.

2018-2019 Investigation of Services delivered for TB by External care system – especially the Private sector (INSTEP) (Collaborator)
I worked on quantitative measure of health seeking pathways and delays, diagnostic and treatment behaviors of private providers and qualitative (or mixed methods) analysis of provider behaviors and the reasons behind them as assessed via direct interviews.

Training Grants and Mentored Trainee Grants

1990-2011 Multidisciplinary AIDS Training Grant
NIH NIAID T32AI007387
Mentor (PI: Martin Hirsch)

The major goal was to provide in depth laboratory experience in a specific research area of virology, immunology, molecular biology, oncology, epidemiology molecular genetics, or molecular therapeutics to selected postdoctoral candidates.

1992-2022 Program for AIDS Clinical Research Training (PACRT)
NIH NIAID T32 AI007433
Mentor (PI: Kenneth Freedberg)
The major goal is to provide training in quantitative research methodologies with a focus on HIV clinical research to pre-doctoral PhD students and physicians at formative stages in their careers.

1998-2020 Epidemiology of Infectious Diseases
NIH NIAID T32 AI007535
Mentor (PI: George Seage)
The major goal is to increase the number of graduates who will be capable of drawing on diverse tools – including sophisticated approaches to causal inference, transmission-dynamic modeling, model fitting, population genomics and phylogenetics – in a knowledgeable way to meet the infectious disease threats of a new generation.

2004-2009 Molecular Approaches for Understanding TB Dynamics
NIH NIAID K08 5K08AI055985
Co-Mentor to Ted Cohen
The major goal of this five-year training program K award focused on the development of new analytic tools to evaluate molecular data from tuberculosis patients.

2009 AMSTH Postdoctoral Fellowship in Tropical Infectious Diseases
Mentor to Karen Jacobson
The major goal was to fund to conduct research focused on infectious diseases of low and low-middle income countries.

2010-2013 Predicting the impact and cost-effectiveness of technical and non-technical approaches to TB control in low and middle income countries
CIHR (Canadian Institute for Health Research) Fellowship MFE106987
Mentor to Olivia Oxlade
The goal was to predict, in 3 low and middle income countries, the epidemiologic impact and cost effectiveness of a technical approach to TB control (using improved diagnostic tests for earlier diagnosis of active TB disease) versus a non-technical population level intervention designed to reduce tobacco use and alcohol consumption.

2010-2014 The Economic Impacts of Community-Based Integrated Health Care Systems in Rural Rwanda
NIH Fogarty K01 TW008773
Mentor to Matthew Bonds

The major goal of this K award was to measure the specific economic consequences of expanded community-based integrated primary healthcare in Rwanda by measuring the partial effects of malnutrition, disease, schooling and socioeconomic status on each other.

2011 Modifiable risk factors for tuberculosis disease in children
Parker B. Francis Fellowship in Pulmonary Research
Mentor to Molly Franke
The major goal was to identify modifiable risk factors for TB in children.

2011-2016 Geospatial Clustering and Molecular and Social Epidemiology of Drug Resistant TB
NIH Fogarty K01 5K01TW009213
Co-Mentor to Karen Jacobson
The major goal of this K award was to estimate the burden of drug resistant TB and assess the heterogeneity of disease burden in different geographic locations, to examine the association of host risk factors and population determinants with regions of high drug resistant TB burden, and to describe the spatial and molecular clustering of strains of drug resistant TB in this province. My role was to mentor Karen Jacobson in research in molecular and social epidemiology of TB.

2012-2013 US-Italy Fulbright Scholarship
Mentor to Anna Odone

2012-2016 The Role of Development Assistance for Health in Reducing Child Mortality
NIH NICHD 4K01HD071929-05
Epidemiology mentor to Chunling Lu
The major goal of this K award was to obtain background knowledge of epidemiology so as to understand the disease profiles of under-five children of different age groups in developing countries.

2013-2014 Controlling Drug Resistant Tuberculosis (TB): A Review of Literature and an Attempt for Designing Innovative Approaches in Indian Setting
Core Fulbright Visiting Scholar Research Grant
Mentor to Sachin Atre

2013-2015 Gene Mutations and Tuberculosis Resistance
American Lung Association Research Award
Mentor to Maha Farhat
The major goal was to investigate the genetic sequences of known and candidate resistant genes for a large panel of TB drugs, to determine which mutations predict the extent of resistance, and if specific combinations of mutations interact to affect this resistance level. The information will be used to guide the development of a much needed rapid diagnostic test for drug resistant TB.

2014 Genetic determinants of drug resistance in mycobacterium tuberculosis
Parker B. Francis Fellowship in Pulmonary Research
Mentor to Maha Farhat
The major goal was to investigate the genetic sequences of known and candidate resistance genes for a large panel of TB drugs to determine which mutations predict the extent of resistance and use this information to guide the development of improved diagnostic tests for resistance.

2014-2017 Integrating Pediatric Care Delivery in Rural Healthcare Systems
NIH NICHD 5DP5OD019894
Mentor (PI: Duncan Maru)
The major goal was to increase the timely engagement in acute care for children to receive evidence-based World Health Organization protocols aimed at reducing child mortality and to implement a Chronic Care Model for pediatric patients under the age of twenty suffering from a chronic disease.

2014-2019 Infectious Disease and Basic Microbiological Mechanisms
NIH NIAID T32 2T32AI007061
Mentor (PI: Marcia Goldberg)
The major goal is to train scientists who have a career goal of solving medically relevant problems and who elect rigorous laboratory or epidemiologic training in any of the Harvard adult infectious disease programs or other Harvard-based institutions participating in this program.

2015-2017 New Tools for the Interpretation of Pathogen Genomic Data with a Focus on Mycobacterium Tuberculosis
NIH Fogarty K01 5K01ES026835
Principal Mentor to Maha Farhat
The major goal of this K award was to develop a web-based public interface to several analysis tools, to develop and study an MTB gene-gene network, and to study the performance of methods in current use for the association of genotype and phenotype in pathogens, and develop a generalizable power calculator for the best performing method.

2016-2017 Genetic Determinants of Drug Resistance in Mycobacterium Tuberculosis
NIH URM Supplement U19AI109755-03S1
PI & Mentor to Ibrahim Diakite (Total direct costs $82,633)
The major goal of this supplement was to develop and validate a prediction model that will define the optimal set of mutations to be assessed to improve the performance of rapid molecular diagnostics.

Report of Local Teaching and Training

Teaching of Students in Courses

Boston University

1998-2001	SPH EB755: Infectious Disease Epidemiology 34 students of public health	Boston University School of Public Health 2.5-hr sessions per week for 15 weeks

Harvard School of Public Health

2001-2003	ID293: Inference in Infectious Disease Epidemiology 15 students of public health	Harvard School of Public Health 4-hr sessions per week for 8 weeks
2001-2004	EPI225: Infectious Disease Dynamics 5 medical students, 50 students of public health	Harvard School of Public Health 4-hr sessions per week for 8 weeks
2002	ID267: Infectious Disease Epidemiology Seminar 2 medical students, 8 students of public health	Harvard School of Public Health 2-hr sessions per week for 16 weeks
2002-2003	ID229: Epidemiology of Infectious Disease Developing Countries 50 students of public health	Harvard School of Public Health 2-hr session
2002	EPI269: Epidemiological Research in Obstetrics and Gynecology 30 advanced students of public health	Harvard School of Public Health 1-hr session
2003-2006	IMI202: Tuberculosis 10 medical students, 10 students of public health	Harvard School of Public Health 2-hr sessions
2003-2004	ID287: Bioterrorism: Public Health Preparedness and Response 30 students of public health	Harvard School of Public Health 1-hr session
2004-2007	EPI285: Infectious Disease Dynamics 50 graduate students of public health	Harvard School of Public Health 5-hr per week for 16 weeks
2008-2015	EPI501: Dynamics of Infectious Diseases 50 graduate students of public health	Harvard School of Public Health 4-hr sessions per week for 8 weeks

2008-2010	GHP539: The Social, Political and Economic Dimensions of Infectious Diseases in Developing Countries 20 medical and graduate students of public health	Harvard School of Public Health 2-hr session
2008	IMI 227: Genetics and Genomics of Infectious Diseases: Tuberculosis, Malaria 25 graduate students of public health	Harvard School of Public Health 2-hr session
2008-2015	ID269: Respiratory Epidemiology 18 medical and graduate students of public health	Harvard School of Public 2-hr sessions
2009-2011	IMI202: Tuberculosis the Host, the Organism and the Global 9 graduate students of public health	Harvard School of Public Health 2-hr session
2015, 2017, 2019	Epi225 Epidemiology of HIV 30 graduate students of public health	Harvard School of Public Health 2-hr session
2016 -	Epi502: Biology and Epidemiology of Antibiotic Resistance 20 graduate students of public health	Harvard School of Public Health 2-hr session

Harvard University/FAS

2004	FAS Freshman Seminar 24p: How Epidemics Happen 12 undergraduate students	Harvard College, Cambridge, MA 3-hr sessions per week for 16 weeks
2005-2006	FAS Freshman Seminar 25m: Epidemics as a Metaphor 12 undergraduate students	Harvard College 2-hr sessions per week for 16 weeks
2006-2007	FAS Freshman Seminar 25m: What Epidemics Mean: Infectious Disease in a Social Context 12 undergraduate students	Harvard College 2-hr sessions per week for 16 weeks

Formal Teaching of Residents, Clinical Fellows and Research Fellows (post-docs)

2003	The Transmission Dynamics of *M. tuberculosis*: Models and Molecular Epidemiology	Research Seminar Department of Epidemiology Harvard School of Public Health

		One-hour lecture
2004	Transmission of TB in the Community Invited Lecture	Infectious Disease Society of America Boston, MA One-hour lecture
2007	Genetic Heterogeneity in *M. tuberculosis*	Department of Genetics and Complex Diseases Harvard School of Public Health One-hour lecture

Clinical Supervisory and Training Responsibilities

1996-2007	Attending and supervision of clinical infectious disease fellows/Massachusetts General Hospital	Daily supervision for 6 weeks per year

Laboratory and Other Research Supervisory and Training Responsibilities

2002-2004	Supervision of Julia E. Aledort, doctoral research fellow/Harvard School of Public Health	Weekly mentorship for 18 months
2002-2006	Supervision of Stephen Resch, doctoral research fellow/Harvard School of Public Health	Weekly mentorship for 18 months
2004-2006	Supervision of Johanna Daily, Master's student /Harvard School of Public Health	Monthly mentorship for 24 months
2007-2008	Supervision of Preetika Muthukrishnan, Master's student/Harvard School of Public Health	Weekly mentorship for 24 months
2009	Supervision of Daniel Pletzer, Undergraduate intern/Upper Austria University of Applied Sciences, Hagenberg, Austria	Daily laboratory mentorship for 3 months
2010	Supervision of Matsie Mphahlele, doctoral candidate at Stellenbosch University, Visiting Fogarty scholar	Weekly mentorship for 3 months

2010	Supervision of Laurence Laser, visiting Master's student from Ecole Polytechnique Federale de Lausanne	Weekly mentorship for 9 months
2018	Supervision of Junkun Ren, Master's student in epidemiology, Harvard T.H. Chan School of Public Health	Mentorship for 3 months

Formally Supervised Trainees and Faculty

1999-2004 Caroline Korves, ScD / Epidemiologist, Analysis Group, Inc.
I was Dr. Korves's doctoral supervisor at the Harvard School of Public Health. Published two research papers together, one in PLoS Medicine and Clinical Infectious Disease.

2001-2006 Theodore Cohen, MD, MPH, DPH / Professor, Department of Epidemiology, School of Public Health, Yale University
I was Dr. Cohen's DPH advisor at the Harvard School of Public Health and his primary mentor on his NIH K08 grant. Published 36 research papers together, including one in Science, one in Nature Medicine, and one in PNAS.

2003-2005 Anson Wright, MSc / WASH Advisor, Millennium Villages Project
I supervised Ms. Wright's master's thesis on preparedness for a *Yersinia pestis* bioterrorism attack.

2004-2006 Kristina Wallengren, PhD, MPH / Executive Director and Founder, THINK (Tuberculosis and HIV Investigative Network)
I was Dr. Wallengren's post-doctoral advisor at Harvard School of Public Health. We published three papers together.

2004-2010 Molly Franke, ScD / Assistant Professor, Department of Global Health and Social Medicine, Harvard Medical School
I was Dr. Franke's doctoral advisor at Harvard School of Public Health and continue to mentor her in her role at HMS. We have published 18 research papers together.

2005-2010 Erin Johnson, PhD / Associate Professor, Department of Biology, John Carroll University
I was Dr. Johnson's post-doctoral advisor at Harvard School of Public Health. Published two papers together in FEMS Immunology and Medical Microbiology and Infection and Immunity.

2005-2009 Hsien-Ho Lin, MD, MPH, ScD / Associate Professor in Epidemiology, Institute of Epidemiology and Preventive Medicine, National Taiwan University College of Public Health

I was Dr. Lin's advisor at Harvard School of Public Health. Published nine research papers together, including in PLoS Medicine, the Lancet, and American Journal Respiratory Critical Care Medicine.

2005-2011 Meghan Baker, MD / Instructor, Department of Population Medicine, Harvard Medical School and Harvard Pilgrim Health Care Institute
I was Dr. Baker's advisor at the Harvard School of Public Health, Boston, MA. We have published five papers together.

2006-2009 Andreas Sandgren, MSc, PhD / Deputy Head, ReAct Europe
I was Dr. Sandgren's post-doctoral research advisor at Harvard School of Public Health. We published three research papers together, including one in PLoS Medicine.

2006-2010 Christie Jeon, MSc, ScD / Assistant Professor, Cedars-Sinai Division of Hematology/Oncology and Department of Epidemiology, UCLA Fielding School of Public Health
I was Dr. Jeon's doctoral advisor at the Harvard School of Public Health. We published ten research papers together including one in PLoS Medicine.

2006-2011 Kathleen Wirth, ScD / Research Scientist, Department of Biostatistics, Harvard School of Public Health
I was Dr. Wirth's doctoral advisor at the Harvard School of Public Health. We published two papers together, including one in Epidemiology.

2006-2008 Caroline Colijn, PhD / Professor, Department of Mathematics, Simon Fraser University
I was Dr. Colijn's post-doctoral advisor at Harvard School of Public Health. Published twelve research papers together, including one in American Journal Respiratory Critical Care Medicine and one in PLoS Computational Biology.

2007-2009 Gape Machao, MSc / Monitoring and Evaluation Officer, UNICEF Botswana
I supervised Mr. Machao's master's thesis on rapid diagnostic testing for TB in Botswana.

2008-2010 Ellen Brooks-Pollock, MSc, PhD / Lecturer, Veterinary Public Health, Bristol Veterinary School
I was Dr. Pollock's post-doctoral research advisor at Harvard School of Public Health. We published two papers together.

2008-2013 Karen Jacobson, MD / Assistant Professor of Medicine, Section of Infectious Diseases, Boston University School of Medicine
I was Dr. Jacobson's research mentor for her infectious disease post-doctoral research fellowship. We published nine papers together.

2008-2010 Tsering Pema Lama, MSc. Postdoctoral Fellow / Consultant, The George Washington University Milken Institute School of Public Health

I supervised Ms. Lama's master's thesis.

2009-2015 Matthew Bonds, PhD / Assistant Professor, Department of Global Health and Social Medicine, Harvard Medical School
I was Dr. Bonds' mentor on his K award on poverty traps and currently mentor him in his role in my department. We have published five papers and two book chapters together.

2009-2012 Razvan Sultana, MD, PhD / Computational Biologist, University of Hawaii John A. Burns School of Medicine
I co-supervised Dr. Sultana's doctoral thesis in Bio-informatics at Boston University on genomic analysis of drug resistant TB. We have published three papers together.

2010-2016 Hanna Guimaraes, MA, PhD / Postdoctoral Researcher, RIVM National Institute for Public Health and the Environment
I was Ms. Guimaraes' doctoral adviser while she conducted research for her degree from Portugal. We published four papers together.

2010-2016 Maha Farhat, MD, MSc / Assistant Professor of Biomedical Informatics, Harvard Medical School
I was Dr. Farhat's postdoctoral mentor and supervised her analysis of whole genome sequence data on *M. tuberculosis* for the identification of novel mutations associated with drug resistance. We have published 12 papers together.

2010 Joanne Salmon, MD, MPH / Clinical Instructor, Division of Infectious Diseases, Department of Medicine, The University of British Columbia
I supervised Dr. Salmon's master's thesis on community health workers and impact on TB treatment outcomes: a multi-country proposal.

2010-2014 Chuan-Chin Huang, MS, ScD / Instructor in Medicine, Harvard Medical School
I was Dr. Huang's doctoral adviser. We have published eight papers together.

2010-2014 Olivia Oxlade, PhD / Epidemiologist and Modeler, McGill International TB Centre
I was Dr. Oxlade's postdoctoral research supervisor in her work on modeling the determinants of TB. We published three papers together.

2010-2016 MaryCatherine Arbour, MD / Assistant Professor of Medicine, Department of Global Health and Social Medicine, Harvard Medical School
I mentored this junior faculty member at the Division of Global Health Equity, Brigham and Women's Hospital in her work on education and health outcomes in a cluster randomized trial of school-based interventions in Santiago, Chile. We published two papers together.

2011 **Devra Barter, MS / Emerging Infections Epidemiologist, Colorado Department of Public Health & Environment**
I co-supervised Ms. Barter's master's thesis on out-of-pocket expenses during TB treatment which is published in BMC Public Health.

2011-2013 Silvan Vesenbeckh, MD / Senior Registrar, Infectious Diseases, Groote Schuur Hospital
I supervised Dr. Vesenbeckh's postdoctoral work on cholera transmission in the DRC and Haiti. We published three papers together.

2011-2015 Philips Loh, MS / Doctoral candidate, Department of Epidemiology, Harvard School of Public Health
I supervised Mr. Loh's master's thesis and served as his doctoral adviser.

2012-2013 Alexis Krumme, MS, ScD / Research Specialist, Division of Pharmacoepidemiology and Pharmacoeconomics, Brigham and Women's Hospital
I supervised Ms. Krumme's master's thesis. We published one paper together.

2012-2015 Xeno Acharya, MPH / Senior Consultant, Healthcare AI, PA Consulting
I was Mr. Acharya's doctoral adviser. We have published one paper.

2013-2014 **Anna Odone, MD, MPH, PhD / Associate Professor of Public Health, Università Vita-Salute San Raffaele**
I was Ms. Odone's postdoctoral research supervisor for her work on socioeconomic risk factors for acquired and primary MDR-TB in Lima, Peru. We published one paper together.

2013-2014 Sachin Atre, PhD / Study Coordinator, Johns Hopkins Center for Clinical Global Health Education
I supervised Dr. Atre's work on MDR-TB management and policy in India, and the effective use of information technology in TB control. We published one paper together.

2013-2015 Emilia Ling, MS / Medical Student, Stanford University
I supervised Ms. Ling's master's thesis at HSPH. We published one paper together.

2013-2016 Assumpta Mukabutera, PhD / Instructor, University of Rwanda School of Public Health
I supervised Dr. Mukabutera's doctoral thesis on rainfall and child health outcomes. We published three papers together.

2014-2016 Rebecca Butler, MS / Biostatistician, Kaiser Permanente
I supervised Ms. Butler's master's thesis.

2014-2016 Gustavo Velasquez, MD, MPH / Research Associate, Department of Global Health and Social Medicine, Harvard Medical School

I supervised Dr. Velasquez's postdoctoral work examining the relationship between phenotypic pyrazinamide resistance and multidrug-resistant tuberculosis (MDR-TB) treatment outcomes. We published four papers together.

2014-2017 Ibrahim Diakite, PhD / Associate Scientist in Modeling & Meta-Analysis, Pharmerit International
I supervised Dr. Diakite's postdoctoral project that aimed to advance the quantitative study of communicable diseases especially the Mycobacterium Tuberculosis by using a combination of different mathematical techniques such as differential equations, stochastic process, branching process, and mathematical game theory. We published two papers together.

2014-2018 Omowunmi Aibana, MD, MPH / Assistant Professor, General Internal Medicine, University of Texas McGovern Medical School
I supervised Dr. Aibana's work on Tuberculosis in Ukraine through a T32 mechanism based at Brown Medical School. We published five papers together.

2014-2019 Eric Mooring, MPhil, ScD / Epidemic Intelligence Service, Centers for Disease Control and Prevention
I was Dr. Mooring's doctoral adviser. We have published three papers together.

2014-2020 Ruoran Li, MPhil / Doctoral Student, Department of Epidemiology, Harvard T.H. Chan School of Public Health. I was Dr. Li's doctoral adviser. We have published one paper together.

2016-2018 Silvia Chiang, MD / Assistant Professor of Pediatrics, Brown Alpert Medical School
I supervised Dr. Chiang in her postdoctoral study of adolescent tuberculosis. We have published two papers together and have one under review.

2017-2018 Katrin Sadigh, MD / Fogarty Global Health Fellow, Harvard T.H. Chan School of Public Health
I supervised Dr. Sadigh in her clinical research as part of the Department of Infectious Disease, Brigham and Women's Hospital/Massachusetts General Hospital combined program.

2017-2019 Taylor Chin, BA / Master's student, Department of Epidemiology, Harvard T.H. Chan School of Public Health
I supervised Ms. Chin's master's thesis.

2017-2019 Tori Cowger, MPH / Doctoral Student, Department of Epidemiology, Harvard T.H. Chan School of Public Health
I was Ms. Cowger's doctoral adviser.

2017- Alexander Chu, MPH / Post-baccalaureate premedical candidate, Harvard Extension School.

2017- Annelies Mesman, PhD / Postdoctoral Research fellow, Department of Global Health and Social Medicine, Harvard Medical School. I supervised Annelies Mesman in her postdoctoral study of tuberculosis. We have published two papers together.

2019 Gerson Galdos Cardenas, PhD / Postdoctoral Research fellow, Department of Global Health and Social Medicine, Harvard Medical School.

2019- Kamela Ng, PhD / Postdoctoral Research fellow, Department of Global Health and Social Medicine, Harvard Medical School.

2019- Qi Tan, MD, PhD / Postdoctoral Research fellow, Department of Global Health and Social Medicine, Harvard Medical School.

Presentations

Invited Presentations and Courses

Local, Regional, National, and International Invited Presentations and Courses

Local Invited Presentations

No presentations below were sponsored by outside entities

2001 Styblo's Rule revisited
Freeman Symposium Research Seminar
Department of Epidemiology
Harvard School of Public Health

2002-2003 Molecular epidemiology of tuberculosis
Invited Lecture
Hot Topics Series
Harvard School of Public Health

2003 The transmission dynamics of *M. Tuberculosis*: Models and Molecular Epidemiology
Research Seminar
Department of Epidemiology
Harvard School of Public Health

2003 Inferring the evolution of *M. Tuberculosis* from comparative genomics
Research Seminar
Infectious Disease Unit
Harvard Medical School

2003 The epidemiology of Severe Acute Respiratory Syndrome (SARS)
Invited lecture

Kennedy School of Government
Harvard University

2003 Transmission dynamics, epidemiology and SARS
Research Seminar
Department of Epidemiology
Harvard School of Public Health

2003 Modeling the molecular epidemiology of TB
Freeman Symposium Research Seminar
Department of Epidemiology
Harvard School of Public Health

2003 Molecular epidemiology and the transmission dynamics of tuberculosis
Research Seminar
The Broad Institute

2004 The epidemiology of SARS
Hot Topic Series
Harvard School of Public Health

2005 Epidemiology of multi-drug resistant tuberculosis
Grand Rounds
Massachusetts General Hospital

2006 Iron metabolism and *M. Tuberculosis*
Research Seminar
The Broad Institute

2006 Natural variation in *M. Tuberculosis*
Research Seminar
The Broad Institute

2006 Avian influenza
Department of Environmental Health
Harvard School of Public Health

2006 Three epidemics and how they happened
Department of Epidemiology Seminar
Harvard School of Public Health

2006 Transmission dynamics of drug sensitive and resistant tuberculosis infectious disease
Research Seminar
Partners Infectious Disease
Boston, MA

2007 Genetic heterogeneity in *M. Tuberculosis*
Department of Genetics and Complex Diseases
Harvard School of Public Health

2007 Epidemiology of HIV and tuberculosis
Department of Epidemiology Seminar
Harvard School of Public Health

2008 A multi-disciplinary approach to MDR and XDR tuberculosis
Department of Epidemiology Seminar Series
Harvard School of Public Health

2008 Making multidisciplinary research work: the example of MDR tuberculosis
Seminar Series
Department of Social Medicine and Health Inequalities
Brigham and Women's Hospital

2008 Conducting research in international settings
Best Practices in International Scientific Collaboration (Panel discussion)
2nd annual New England Tuberculosis Retreat
Harvard Initiative for Global Health
Harvard Medical School

2009 Genomic epidemiology of MDR and XDR tuberculosis
The Broad Institute

2009 A multi-disciplinary approach to XDR tuberculosis
Grand Rounds
Department of Medicine
Brigham and Women's Hospital

2009 Social justice and the effort to address MDR TB
Symposium on an Idea of Justice
Harvard University and the China Research Council

2010 The evolution of drug resistant tuberculosis
Grand Rounds
Department of Medicine
Massachusetts General Hospital

2010 Overview of Murray research team
Freeman Symposium Research Seminar
Department of Epidemiology
Harvard School of Public Health

2011 Deans' Research Update
Harvard School of Public Health

2011 Innovation in global health
Massachusetts General Hospital Department of Medicine Bicentennial Reunion
Department of Medicine
Massachusetts General Hospital

2013 TB in the 21st century: the convergence of the infectious and metabolic diseases
Seventh Annual New England Tuberculosis Symposium
The Broad Institute

2014 Ebola and the research equity agenda
Global Health Advisory Council
Harvard Medical School
Boston, MA

2015 Ebola Update
Global Health Advisory Council
Harvard Medical School
Boston, MA

2015 HIV and TB co-infection
Harvard T.H. Chan School of Public Health
Boston, MA

2015 Burke Global Health Fellowship Symposium
Harvard Global Health Institute
Cambridge, MA

2017 Host and bacterial determinants of TB infection and disease: a longitudinal cohort study
Spring Seminar
Center for Communicable Disease Dynamics
Harvard T.H. Chan School of Public Health, Boston, MA

2017 Host and bacterial determinants of TB infection and disease: insights from a large cohort study
IDMP Seminar
Broad Institute of MIT and Harvard, Cambridge, MA

2017 Tuberculosis and the vitamin A connection
Talks at 12
Harvard Medical School

2018 How to write an NIH grant
Training to Teachers Mongolia

Harvard Medical School

2020 SARS-CoV-2: Assessing the risks
Environmental Health Risk: Analysis and Applications (RISK0320)
Harvard T.H. Chan School of Public Health

2020 The epidemiology of COVID-19: Evaluation of Treatment and Response
GHSM Seminar - Social Medicine: Response to COVID-19
Harvard Medical School

2020 The Epidemiology of COVID-19
MassCPR webinar

2020 BCG and innate immune responses
Pathogenesis Working group
MassCPR webinar

2020 The transmission dynamics of COVID-19
BCMP Seminar
Harvard Medical School

Regional

No presentations below were sponsored by outside entities

2001 Genetics and phenotypic variability within *M. Tuberculosis*
Invited lecture
Boston University

2001 Problems in the molecular epidemiology of tuberculosis
Research Seminar
Massachusetts State Laboratory Institute (MSLI), Boston, MA

2006 Three epidemics
Kay Stratton Lecture
Massachusetts Institute of Technology, Cambridge, MA

2015 Converging epidemics: tuberculosis and diabetes
Oxford Immunotec
Marlborough, MA

National

No presentations below were sponsored by outside entities

2004 Transmission of TB in the community
Infectious Disease Society of America, Boston, MA

2006 Modeling MDR tuberculosis
National Partners Meeting on MDR Tuberculosis, Atlanta, GA

2006 Transmission dynamics of Drug Resistant tuberculosis
Interscience Conference on Antimicrobial Agents and Chemotherapy, San Francisco, CA

2008 Host iron metabolism genes
Workshop on Biofilms, Iron and Drug Refractory TB
Colorado State University, Fort Collins, CO

2008 The impact of strains diversity and mechanisms of strains competition on the Potential Performance of New TB Vaccines
Microbial Diseases Lecture Series
Yale School of Public Health, Hartford, CT

2008 The role of mathematical modeling in evaluating interventions to control epidemics: the example of tuberculosis
Howard Hughes Medical Institute
California Institute of Technology, Pasadena, CA

2008 An interdisciplinary approach to extensively drug resistant tuberculosis
Howard Hughes Medical Institute
California Institute of Technology, Pasadena, CA

2008 Number of MDR-TB and XDR-TB patients receiving treatment today: Successes/Failures/Consequences
Forum on Drug Discovery, Development and Translation
Institute of Medicine of the National Academies, Washington, DC

2009 The evolution of XDR-TB in *M. tuberculosis*
Seminar Series Biology Department
Williams College, Williamstown, MA

2009 The evolution of XDR-TB in *M. tuberculosis*: a multidisciplinary approach
Grand Rounds Presentation
Division of Infectious Diseases
Hennepin County Medical Center, Minneapolis, MN

2009 Mathematical modeling
Infectious Diseases Clinical Cases Conference
Division of Infectious Diseases
Hennepin County Medical Center, Minneapolis, MN

2009 The evolution of XDR-TB: a multidisciplinary approach
2009 National TB Conference-TB Elimination-"It Takes a Village"
Centers for Disease Control and Prevention, Atlanta, GA

2009 The Gates Project overview
Mycobacteriology Laboratory Branch (MLB)
Division of Tuberculosis Elimination Seminar
Centers for Disease Control and Prevention, Atlanta, GA

2010 Social, economic and biological determinants of tuberculosis
Taskforce for Disease Eradication
Carter Center
Atlanta, Georgia

2010 Estimating the impact of social and biological determinants on TB and modeling their modification
Texas School of Public Health
Brownsville, Texas

2010 The evolution of XDR tuberculosis
Mary Hitchcock Hospital
Hanover, New Hampshire

2011 Molecular methods to detect drug resistance in *M. tuberculosis*
Workshop on TB and HIV Diagnostics in Adult and Pediatric Populations
National Institutes of Health
Washington, DC

2011 Understanding the transmission dynamics of drug resistant tuberculosis: a multidisciplinary approach
Annual Biomedical Research Conference for Minority Students
St. Louis, Missouri

2012 Evolution of drug resistance
World TB Day Symposium
Boston University
Boston, Massachusetts

2012 High throughput sequencing of drug resistance targets for Mycobacterium tuberculosis
National Institute of Allergy and Infectious Diseases
Sponsored meeting
Washington, DC

2013 Genetic determinants of drug resistance in *Mtb*
World TB Day Symposium
Weill Cornell Medical College
New York City, New York

2015 Tuberculosis and diabetes
The Comstock Lecture
Johns Hopkins School of Public Health
Baltimore, Maryland

2016 Tuberculosis and diabetes
20th Annual Conference of Union-North America Region/National TB Controllers Association Joint Meeting
Denver, Colorado

2017 Risk factors for TB disease progression: evidence from a cohort study in Peru
9th Annual CEND (Center for Emerging and Neglected Diseases) Symposium: Deconstructing TB: Insights from Fundamental Research
University of California, Berkeley, California

2017 Public health and the environment: interdisciplinary research and emerging infectious disease
Ecology & Evolution of Infectious Diseases
UC Santa Barbara, California

2018 Women in science
2018 Women in Science Symposium
Colorado State University
Fort Collins, Colorado

2019 Who gets TB infection and disease in Lima, Peru
Epidemiology Grand Rounds
Columbia University Mailman School of Public Health
New York, New York

2019 Who gets TB infection and disease in Lima, Peru
UPGG Tuesday Seminar Series
Duke University
Durham, North Carolina

International

No presentations below were sponsored by outside entities

2001 Determinants of cluster distribution in *M. tuberculosis*
Research Seminar
University of Warwick, Coventry, United Kingdom

2002 Pathogenesis of tuberculosis
Invited Lecture
Peruvian Thoracic Society, Lima, Peru

2002 Problems in the molecular epidemiology of tuberculosis
Research Seminar
Karolinski Institutet, Stockholm, Sweden

2003 The fitness of MDR-TB: what do we know
Invited Lecture
World Health Organization, Tallin, Estonia

2004 Molecular epidemiology of TB in Sverdlosk, Russia
Invited Lecture
International Union Against Tuberculosis and Lung Disease (IUTLD) Meeting
Moscow, Russia

2005 The fitness of MDR-TB strains
Invited Lecture
Desmond Tutu Center for Tuberculosis Research
University of Stellenbosch, Matieland, South Africa

2005 The current and future status of Multi-Drug Resistant TB
Invited Lecture
Novartis Symposium on TB Drug Development, Bagamoyo, Tanzania

2006 XDR-TB surveillance
Task Force on XDR-TB
World Health Organization, Geneva, Switzerland

2007 Mathematical models of population effects of potential TB vaccines
Keystone Symposium of Challenges of Global Vaccine Development
Cape Town, South Africa

2007 Modeling vaccine effects
Modeling Symposium at the 38th Union World Conference on Lung Health
Cape Town, South Africa

2007 Genomic epidemiology of infectious diseases: a new science
US-Japan Meeting
Hainan, China

2008 The molecular evolution of extensively drug resistant tuberculosis
Keystone Symposium of Pathogenesis and Control of Emerging
Infections and Drug-Resistant Organisms

Bangkok, Thailand

2008 TB Drug Resistance Mutation Database
39th Union World Conference 2008
International Union of TB and Lung Disease
Paris, France

2009 The evolution of multi-drug resistance in *M. tuberculosis*
Engineering and Physical Sciences Research Council Workshop
on the Evolution of Antibiotic Resistance
Imperial College, London, England

2009 The evolution of multi-drug resistance in *M. tuberculosis*
School of Biosciences Seminar Series
University of Birmingham, Birmingham, England

2009 Diagnosis of drug resistant TB
Fondation Mérieux International Scientific Conference on Latest Approaches to HIV
Infection Management: A Focus on HIV, TB and HIV/Hepatitis Co-Infection
New Delhi, India

2009 Tuberculosis and diabetes: interactions between two epidemics
TB/DM Expert Meeting
International Union of TB and Lung Disease
Paris, France

2009 Differences between epidemiology of TB in rich and poor countries
Union World Conference of Lung Health
Cancun, Mexico

2009 Modeling the potential impact of changing risk factors and social determinants
Union World Conference of Lung Health
Cancun, Mexico

2010 The evolution of drug resistance in TB
Ecole Polytechnique Federale de Lausanne
Lausanne, Switzerland

2010 Identification of drug resistance mutations in *M. tuberculosis*
Fondation Mérieux
Annecy, France

2010 Guidelines for management of tuberculosis and diabetes
World Health Organization
Geneva, Switzerland

2010 Data for developing diagnostics for MDR TB
Christian Medical College
Vellore, India

2010 Beyond labs and pills for improved tuberculosis control: what role for TB programmes?
41st Union World Conference on Lung Health
Berlin, Germany

2011 Iron transport polymorphisms and TB susceptibility
Ecole Polytechnique Federale de Lausanne
Lausanne, Switzerland

2011 Evaluating health interventions using DHS oversamples
Doris Duke Charitable Foundation Population Health Implementation Training Partnership Grantee Meeting
Ifakara, Tanzania

2012 Overview: Transmission dynamics and epidemiology of drug resistant TB
Lima, Peru

2012 The transmission dynamics of drug resistant TB
Harvard China Fund
Shanghai, China

2012 Understanding the epidemic dynamics of drug resistant TB
Keystone Symposia Conference – Drug Resistance and Persistence in Tuberculosis
Kampala, Uganda

2012 The social, environmental and biologic determinants of tuberculosis
TB Day in Braga: From the hospital to the bench and back.
Braga, Portugal

2012 Studying the link between nutrition and TB Risk: problems and strategies
International Conference of the Union for TB and Lung Disease
Kuala Lumpur, Malaysia

2012 TB and diabetes: what we know, what we don't know
International Conference of the Union for TB and Lung Disease
Kuala Lumpur, Malaysia

2013 Genetic diversity of DR TB: implication for future diagnostics
Institute of Medicine and Chinese Academy of Sciences Workshop

The Global Crisis of Drug-Resistant Tuberculosis and the Leadership of the BRICS Countries: Challenges and Opportunities
Beijing, China

2013 Evolution of drug-resistance in TB genomes
International Conference of the Union for TB and Lung Disease
Paris, France

2014 The genetics and pathogenesis of MDR and XDR TB drug resistance
Conference on Retroviruses and Opportunistic Infections (CROI)
Boston, MA

2014 Genetic basis for transmission of MDR-TB
9th International Conference on the Pathogenesis of Mycobacterial Infections
Stockholm, Sweden

2015 Issues in the management and prevention of drug resistant and sensitive TB
Invited Lecture
Bogomolets National Medical University
Kiev, Ukraine

2015 TB and diabetes mellitus outcomes
19th Annual Conference of the Union-North America Region
Vancouver, BC, Canada

2015 The transmissibility of drug resistant TB
RePort Consortium Meeting
Boston University
Boston, Massachusetts

2015 The TB drug resistance database
46th Union World Conference on Lung Health
Cape Town, South Africa

2016 Diabetes and environmental co-morbidities with TB
Keystone Symposia Conference - Tuberculosis Co-Morbidities and Immunopathogenesis
Keystone, Colorado

2016 Enabling next generation whole genome sequencing readouts directly from sputum samples and in the clinic: hype or hope?
47th Union World Conference on Lung Health
Liverpool, United Kingdom

2017 Infectiousness and transmission of tuberculosis
American Thoracic Society 2017 International Meeting
Washington, DC

2017 Recent insights in the meaning of latency in tuberculosis
30th Annual Doctor Dorothy Wiselberg Seminar
McGill University
Montreal, QC, Canada

2017 Next generation whole genome sequencing for tuberculosis: ready for clinical practice?
48th Union World Conference on Lung Health
Guadalajara, Mexico

2017 Estimating the adolescent tuberculosis burden in the 30 high-TB burden countries
48th Union World Conference on Lung Health
Guadalajara, Mexico

2017 Insights into TB from a longitudinal cohort study in Lima, Peru
National TB program
Lima, Peru

2018 Insights into TB from a longitudinal cohort study in Lima, Peru
Otago University
Dunedin, New Zealand

2018 Grant Writing
Mongolian National University of Medical Sciences
Ulaanbaatar, Mongolia

2019 Bacterial determinants of TB progression
50th Union World Conference on Lung Health
Hyderabad, India

2019 Genetic variations of mycobacterium tuberculosis that are associated with tuberculosis transmission
50th Union World Conference on Lung Health
Hyderabad, India

2020 The COVID-19 Research Agenda
Partners in Health

2020 SARS-CoV-2
Partners in Health

Report of Clinical Activities and Innovations

Current Licensure and Certification

1996	Licensed in Medicine in Massachusetts
1996	Board Certified in Internal Medicine
1997	Board Certified in Infectious Disease

Practice Activities

1998 – 2007	Attending Physician	Infectious Disease Consult Services, MGH, Boston, MA	3-5 new consults, 20-30 follow-ups per day 3-6 weeks per year

The unit provides consults on infectious disease issues to all medical, surgical and other specialty wards. I saw patients referred to the specialty unit in conjunction with a team that includes an infectious disease fellow and rotating medical students and residents. In addition to this bedside clinical teaching, I also participated in weekly clinical conferences and seminars that are designed to maximize teaching.

Report of Scholarship

Peer reviewed publications in print or other media

Research investigations

1. Murray MJ, Murray NJ, Murray AB, **Murray MB**. Refeeding-malaria and hyperferraemia. Lancet 1975;1:653-4.
2. Murray MJ, Murray AB, **Murray MB**, Murray CJ. Somali food shelters in the Ogaden famine and their impact on health. Lancet 1976 Jun 12;1:1283-5.
3. Murray MJ, Murray AB, **Murray MB**, Murray CJ. Parotid enlargement, forehead edema, and suppression of malaria as nutritional consequences of ascariasis. Am J Clin Nutr. 1977 Dec;30(12):2117-21.
4. Murray MJ, Murray AB, Murray NJ, **Murray MB**. Diet and cerebral malaria: the effect of famine and refeeding. Am J Clin Nutr. 1978 Jan;31(1):57-61.
5. Murray MJ, Murray AB, Murray NJ, **Murray MB**. Serum cholesterol, triglycerides and heart disease of nomadic and sedentary tribesmen consuming isoenergetic diets of high and low fat content. Br J Nutr. 1978 Jan;39(1):159-63.

6. Murray MJ, Murray AB, Murray NJ, **Murray MB**. The effect of iron status of Nigerien mothers on that of their infants at birth and 6 months, and on the concentration of Fe in breast milk. Br J Nutr. 1978 May;39(3):627-30.
7. Murray MJ, Murray AB, **Murray MB**, Murray CJ. The adverse effect of iron repletion on the course of certain infections. Br Med J 1978 Oct 21;2:1113-5.
8. Murray MJ, Murray AB, Murray NJ, **Murray MB**, Murray CJ. Molluscum contagiosum and herpes simplex in Maasai pastoralists; refeeding activation of virus infection following famine? Trans R Soc Trop Med Hyg. 1980;74(3):371-4.
9. Murray JM, Murray AB, **Murray MB**, Murray CJ. Rarity of planar warts in Cushite nomads: antiviral effect of milk? Lancet. 1980 Jul 19;2(8186):143-4.
10. Murray MJ, Murray AB, Murray NJ, **Murray MB**. Infections during severe primary undernutrition and subsequent refeeding: paradoxical findings. Aust N Z J Med. 1995 Oct;25(5):507-11.
11. **Murray M**, Rasmussen Z. Measles Outbreak in a Northern Pakistani village: epidemiology and vaccine effectiveness. Am J Epidemiology 2000;1:811-9.
12. Piatek A, Telenti A, **Murray M**, El-Hajj H, Jacobs WR Jr, Kramer FR, Alland D. Genotypic analysis of *M. tuberculosis* in two distinct populations using molecular beacons: implications for rapid susceptibility testing. Antimicrob Agents Chemother 2000;1:103-10.
13. **Murray MB**, Determinants of cluster distribution in the molecular epidemiology of tuberculosis. Proc Natl Acad Sci U S A 2002 Feb; 99:1538-43.
14. **Murray MB**, Alland D. Methodological problems in the molecular epidemiology of tuberculosis. Am. J. Epidemiology 2002;155: 565-71.
15. **Murray MB**. Sampling bias in the molecular epidemiology of tuberculosis. Emerg Infect Dis 2002 Apr; 4:363-9.
16. Hughes A, Friedman R, **Murray M**. Genomewide pattern of synonymous nucleotide substitution in two complete genomes of *Mycobacterium tuberculosis.* Emerg Infect Dis 2002 Nov;8:1342-6.
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Thesis

Murray, MB. Problems in the Molecular Epidemiology of Tuberculosis [Dissertation]. Boston (MA): Harvard School of Public Health; 2001.

Narrative

To date, my career has focused on two main areas: advancing progress in tuberculosis management and control and developing research capacity in low and middle-income countries.

My work on tuberculosis has shifted over the past twenty years from a focus on dynamical modeling of TB epidemics to field studies on the bacterial and host determinants of TB infection and disease. Between 2008-2013, I led a multi-disciplinary consortium that studied the impact of drug resistance of the transmission dynamics of tuberculosis in Lima, Peru. This project followed over 18000 people for TB-associated outcomes and has generated data that has allowed my team to also address a range of host and environmental factors that contribute to the transmission and disease burden of TB. More recently, our work in this area has centered on the links between host metabolic and immune function as determinants of the outcome of TB infection. This work, which is funded through an NIH consortium grant which I co-lead with Dr. Branch Moody, is another multi-disciplinary collaboration, this time among immunologists, epidemiologists, geneticists and veterinary pathologists.

My work on drug resistant tuberculosis has also led me to use targeted and whole genome sequencing to study "genomic epidemiology" and to elucidate the genetic basis of drug resistance phenotypes. To date, we have sequenced over 1500 TB strains and have created an innovative data interface tool that allows us to use whole genome data in epidemiologic studies. Currently, we are funded by NIH to identify, collect, archive, sequence and analyze the drug resistance genes in *M. tuberculosis* strains from around the world. These data are then passed to our collaborators who attempt to validate our findings by generating and phenotyping *Mtb* variants and to our industry partners who are developing point of care diagnostic tests to detect drug resistance. I am the PI of this collaborative project which is funded through an NIH Center for Excellence in Translational Research.

In addition to my roles on my grant-funded projects, I am the research director for the Division of Global Health Equity in the Department of Medicine at the Brigham and Women's Hospital and the non-governmental organization, Partners In Health (PIH). In that capacity, I support the research mission of the Global Health Delivery Partnership by building research infrastructure and mentoring junior faculty interested in research careers. At HMS, I lead the Department of Global Health and Social Medicine's "research core," a team of eight epidemiologists, biostatisticians and programmers in the task of identifying and developing research opportunities in affiliation with PIH and other NGO's clinical field sites. Much of this work focuses on developing methods to evaluate the health interventions implemented in these sites and in designing and carrying out studies to conduct such evaluations. Increasingly, our mission has encompassed the training and development of independent researchers from the countries in which we work.

Almost all my academic work has been conducted in the context of training graduate students and post-doctoral fellows. I have directly supervised 39 graduate students or post-doctoral fellows, almost all of whom have published with me. Fourteen of my former trainees have gone

on to tenure track faculty positions and six have joined international and non-governmental organizations focused on global health. Among many committee assignments, I am particularly proud of my contribution to the Task Force on Women in Science and Engineering which made recommendations that I believe have improved the working lives of many women in science at Harvard. I have served on the Human Subjects Committee at HSPH, co-chaired the Community Engagement Mission of the Strategic Leadership Team at the Brigham and Women's Hospital and led a number of junior and senior faculty searches.